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PHYSIOLOGY OF THE LARYNX. A RESUME AND DISCUSSION OF THE LITERATURE FOR 1940.*

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Chevalier Jackson, in a tremendously important contribution upon "Myasthenia Laryngis,"[1] presents as part of his discussion a number of most interesting physiological observations. His subtitle, "Observations on the Larynx as an Air Column Instrument," tells in a few words the basic physiological principles upon which the production of the vocal sounds depends. Because of the importance of this article, I shall dwell upon it at some length. Dr. Jackson points out that because so much of our theory upon this subject has been imaginative, it is necessary to start with the fundamentals of phonetics and build up a new and correct conception of the phonation mechanism of the larynx. There can be no doubt that many misconceptions are prevalent and this illustrious author cites them as follows:

1. The assumption that laryngeal phonation is analogous to the tone production of a stringed musical instrument.

[*Actually and in fact, at the present time very few authorities hold it to be true that the production of tones by the larynx is entirely analogous to the physical principles involved in the action of a stringed instrument. Guthrie, Curry, Negus, Russell, Paget, Wessely and others, all of*

*For reasons beyond my control, this review is not complete. To those authors, especially abroad, whose articles, published late in 1940, are not included, I extend my sincere apologies.

†Inclusions in brackets represent my own personal views upon the point at hand.

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whom must be considered as foremost authorities upon the subject, have, as I pointed out in previous reviews,[2,3] leaned away from this point of view and deny, as does Jackson, that any similarity exists. Perhaps I personally, more than anyone else, have been guilty of making comparisons between the two, although I have at no time contended that the sound-producing larynx acted entirely as a stringed instrument. But there are, beyond all question of a doubt, striking similarities. For example, in the vibrating string the mass of the vibrating body affects the pitch of the tone produced. A thicker or heavier violin string, other factors such as length and tension being equal, produces a lower tone than a thinner string. So it is in the larynx, as Hartman and Wullstein[4] have demonstrated by rather convincing experiments. Thus, a vibrating cord of less bulk produces a higher tone than the same cord in a thickened state. But the similarity does not end there. If the tension of a vibrating string is increased the pitch is elevated. No one can deny this. A comparable situation very likely exists in the vocal cords, as demonstrated by the Bell Telephone motion pictures, so that there may be some reason to question Jackson, who, in his current article, cites as a second misconception "the assumption that changes in pitch are due to stretching of the vocal cords," which, if this is a misconception, would strongly mitigate against the "string theory." Strictly speaking, this objection may be true, but if such elevation of the pitch of laryngeal tone is not actually CAUSED by stretching and increase of tension of the vocal cords, it is none the less an absolute certainty that such elevations of pitch are, in part of the scale at least, accompanied by if not actually caused by, stretching of the vocal cords. It is only necessary to view the Bell Telephone high speed pictures of the larynx to be entirely convinced of this. A third point in which the vibrating vocal cord seemingly produces changes in pitch in a similar manner to that of a string instrument is the example of "falsetto" tones. In this mechanism it is generally agreed, since the days of Thomas French,[5] who probably was the first to demonstrate the phenomenon, that increasingly higher tones are produced by a gradually increasing FUNCTIONAL foreshortening produced by the complete approximation of a posterior segment of one cord to the other, leaving, as the scale ascends, an increasingly shorter segment of cord free to vibrate. This is entirely com-

parable to the elevation of pitch produced by "fingering" a violin string, thereby similarly shortening the length free to vibrate. In the case, therefore, of both the violin string and the "falsetto" (or dampening mechanism[7]) of the larynx a shorter vibrating segment produces a higher tone, so that a decided similarity does exist between the two. It must be pointed out, however, that as this dampening of one cord by the other takes place, the hiatus between the cords becomes increasingly smaller in area which may, in some measure at least, account for the elevation of pitch.[7] I cite these observations only in order that discussion upon the subject may remain open, rather than in an effort to convince anyone of the truth or error of one theory as against the other. I personally do not know which is correct. Very likely neither the "air-column" nor the "vibrating string" theory are entirely correct since actually it is unlikely that only one mechanism of pitch variation is present, to the exclusion of all others. It would seem to me that the question is still very much unsettled, and awaits its solution in the laboratory through the combined efforts of the physicist, the physiologist and the laryngologist. — J. P.]

Jackson cites further misconceptions:

2. The assumption that the impact of the vibrating cord sets the air in vibration.

3. The assumption that the cricothyroid muscles create tension.

[*I disagree that this is a misconception, and believe that they do create tension by a process of causing elongation of the vocal cords. By those who deny that "stretching" or "elongation" of the vocal cords takes place (Guthrie[5] and Curry,[24] for example), Dr. Jackson's stand upon this point will meet with instant favor. I feel, through observation of the Bell Telephone pictures, that very appreciable elongation, and resulting increase of tension, does take place and is an important mechanism in causing pitch elevation. Since I am rather convinced of this, it is necessary for me to explain the anatomical mechanism by means of which such elongation occurs. I can think of no other way except through the tilting upward in a rotary direction, of the thyroid cartilage and the attached anterior extremities of the vocal cords, by*

action of the cricothyroidii. That tilting upward of the vocal cords does take place is well proven by Canuyt and Gunsett[21] *in an article to be reviewed in later pages. Accordingly, it would seem that these latter muscles do play some rôle in creating tension. An article under my name is about to be published, in which I have dwelt at some length upon this particular point. The importance of this muscle as a creator of tension finds further support in the writings of Beyer upon cricothyroid muscle paralyses*[13] *and those of Redoglia.*[29] *Both of these articles describe the loss of tension occurring after paralysis of the superior laryngeal nerve which supplies the cricothyroid muscle. Both are reviewed at greater length in subsequent pages.* — J. P.]

Dr. Jackson continues to enumerate the popular misconceptions as follows:

4. The assumption that the thyroarytenoid muscles are either laxors or tensors of the vocal cords.

5. Lack of realization of the enormous load of work that is placed upon the thyroarytenoid muscles. All laryngeal muscles are given equal consideration, as though each shared equally in carrying the burden.

6. Misunderstanding of the mechanics of changes in pitch.

7. The assumption that the larynx alone is concerned in changes of pitch.

8. The assumption that the tracheobronchial air column is not concerned in phonation otherwise than in supplying air and air-pressure.

Dr. Jackson continues by deploring the use of the misleading term "vocal cords" since this implies an incorrect analogy to strings stretched for pitch. They should really be considered as callous edges of the thyroarytenoideus muscles. The use of the word "cord" is due to the illusion of the shadow created by the overhanging ventricular band.

The elements in the mechanism of the human voice are grouped by the author under the following heads:

1. The vocal cords.

2. The infraglottic or tracheobronchial air-column.

3. The supraglottic air-column.

4. The molds of speech.

Sounds produced by the vocal cords and emanating from the larynx are modified by two distinct mechanisms, the resonators and the molds of speech, which cut up and form the laryngeal sound into words. The chief factor in producing pitch changes is the accurately controlled forming of the vocal cords under the action of the thyroarytenoid muscle. Supplementary factors are changes in *a.* the size and shape of the glottis, *b.* the dimensions of the infraglottic air-column, *c.* the size and shape of the supraglottic air-column, and *d.* the tracheal air-pressure.

The larynx is essentially an air-column instrument. The loudness of the human voice would be impossible if it depended solely on the impact of the vibrating cords.

[*Although I cannot recall the source, there very clearly stands out in my mind the memory of having read certain reported experiences which completely prove this point as made by Dr. Jackson. An individual with a traumatic fistula into the larynx above the vocal cords had a voice very much more feeble than prior to his injury. Following plastic surgery and closure of the fistula, the voice regained its original loudness.* — J. P.]

Dr. Jackson continues by pointing out that the action of the glottis is to cut the expiratory blast into little puffs, which in turn set into vibration the entire resonating air-column above and below the glottis. The frequency of cutting of the passing air coincides with the vibratory frequency relative to the unit of time, or, in a word, pitch.

[*This description is very convincingly supported by Carhart's experiments,*[31] *also reviewed at length in later pages.* — J. P.]

Changes in the glottic aperture necessary to change the pitch of the fundamental note are produced by the thyroarytenoid muscles, which require rigid fixation at their bases and the taking up of slack in the cords. These two actions are the function of the cricoarytenoid postici, the cricothyroidii, the cricopharyngeus, and to a lesser extent all the

anterior muscles of the neck. The action of the thyroarytenoidii, the cricoarytenoidii laterales and the arytenoideus, if unresisted, would have an orbicular effect on the glottic aperture, against which "guying" is necessary. This guying is not a stretching of the cord but rather a preparation to resist counterpull. This guying and fixing immediately precedes the orbicular action. It is to be especially noted that these terms, orbicular and divergent (or guying), must not be taken as synonymous with adductor and abductor.

Insofar as the human air-column is concerned, it extends from the lips and nostrils to a level at least as low as the orifices of the bronchi of the lower lobes. It is at least 36 cm. in length and its vibrations extend even below this level. The dimensions of the tracheobronchial tree are constantly changing during the respiratory cycle, which must have an important bearing on human phonetics. The influence of this is chiefly on quality of tone and, to a slight degree, on intensity, and it is not altogether without influence on pitch, as indicated by the following laws of physics:

1. The pitch of air vibrations in a tube varies inversely as the length of the tube.

2. The pitch may be somewhat affected by changes in the diameter of the tube.

3. An open tube gives a fundamental tone an octave higher than that of a tube of the same length with one end stopped.

The supraglottic air-column is an open tube; the infraglottic air-column is a tube stopped at its lower end, and the relation of each of these to the source of the fundamental note is different, so that each requires separate consideration. The infraglottic air-column acts as a resonator and as such it reinforces the laryngeally produced sound, and by reinforcing the fundamental note or the partial notes it may influence pitch to some extent. Of course, the pressure of the infraglottic air-column is a large factor in the production of pitch. The pressure increases progressively as the pitch is elevated.

Insofar as the supraglottic air-column is concerned, this becomes shortened for high-pitched notes to an extent that is small compared to inanimate air-column instruments.

Although the thyroarytenoidii with the assistance of the guying muscles do most of the work in varying pitch, it is erroneous to assume that all changes in pitch are produced by the larynx alone, but that substantial aid is received from the changes in the supraglottic and infraglottic air-columns.

At this point Dr. Jackson inserts a paragraph which seems to me to represent the entire sum and substance of the question at hand and which, from the standpoint of logic, appeals to me as more accurately explaining the true state of affairs than any theory yet suggested. He says ". . . I believe that the pitch of the cords acting as reeds is different from the tubal pitch of the whole human air-column, and the pitch of the two sounded simultaneously is different from the pitch of either sounded alone." This finds strong support in Hala's work,[28] in which he concludes from oscillographs that the laryngeal voice is not a simple sound but contains numerous harmonics. The vocal resonances and component sounds do not coincide mathematically as in reality they cross each other, thus exercising a reciprocal influence and making it impossible to distinguish them.

[*Thus it is pointed out by Jackson*[1] *that the tones of the human voice represent a combination of tones produced at the vocal cords, and a second series of tones produced by movement of air within the various human respiratory air channels. It would seem to me that this theory cannot fail to be looked upon favorably by all students of the subject. There is no reason why the movement of air in the human air tubes should not produce tones just as it does in the pipe of an organ or in a test-tube when we blow across its mouth. Furthermore, there is no reason to doubt that tones so produced would be ineffective in controlling, to some extent at least, the final pitch of the tone as it passes out of the mouth.* — J. P.]

Dr. Jackson ascribes an important function to the epiglottis in the control of pitch. Its presence and controlled changes in shape have an important part in the effect of the supraglottic air-column which in itself is an important factor in establishing pitch variations.

In low tones the larynx descends and the hyoid moves forward and downward. In high tones the entire larynx rises

and moves forward. The hyoid rises but less than the larynx, so that the thyroid cartilage approaches the hyoid bone. In addition there is a modification of the size and shape of the supraglottic air spaces and their contained structures, by the action of the extrinsic muscles in and about the mouth and pharynx.

The mechanism of the production of laryngeal sound is compared with the production of tones by various musical instruments and likened very closely to that of the cornetist.

[*This is also mentioned in an article by Guthrie*[2] *as having been first described by Sir Richard Paget. In this connection it is interesting to note that if a motion picture projector is placed on its side so that slow motion photographs of the larynx are projected to lie horizontally on the screen instead of vertically, and thereby come to lie in the same position as we are accustomed to visualize human lips, the similarity of their action during phonation to that of a trumpeter's or bugler's lips cannot be denied by even the most skeptical.* — J. P.]

Concerning the matter of "puff formation" of the air column as it escapes from the larynx, as described by Dr. Jackson, there is, as I have already mentioned, a very interesting article by Raymond Carhart, describing experiments which completely support Jackson's views. In a discussion of air flow through the larynx, Carhart[31] mentions the several theories as to how the sound waves of the laryngeal note are initiated. Some writers claim that vocal fold vibrations are transmitted directly to the surrounding air; others say that periodic changes in the glottic slit area release puffs of air which in turn initiate the sound waves. Recently British physicists (Richardson and Wood) have put forward the hypothesis that laryngeal sound is the result (at least in part) of eddies formed in the air-stream passing through the larynx. In other words, the vocal cords induce "vortex formation" in the stream of air as it passes through the glottal slit.

[*Webster's International Dictionary defines "vortex" as a "mass of fluid having a whirling or circular motion tending to form a cavity or vacuum in the center of the circle and to*

draw towards this, bodies subject to its action; a whirlpool or eddy." — J. P.]

Carhart's paper is an examination of this hypothesis and a description of experimentation testing its validity.

The author points out that, generally speaking, the formation of eddies in an air stream does or may produce musical notes, as in the case of the friction caused by an air stream rushing past a cylindrical wire. Two vortices or eddies revolving in opposite directions are set up on the lee side of the wire. These vortices alternately break away from the wire and this breaking away process at regular intervals sets the wire into vibration, producing sound.

If this principle applies to sound production in the human larynx then, Carhart points out, air flow through the larynx must possess the characteristics typical of vortex tones. In order to test this upon the living larynx the breath stream would have to be rendered visible and from the proper angle. Since this is impossible, the author has substituted studies of air flow upon a "cushion-pipe model larynx, which he admits is not a perfect functional counterpart of the human larynx, but since the model and the normal living larynx are analogous in the functional characteristics which determine air-flow patterns, this cushion-pipe model is adequate for the present study. Two inflatable rubber wall vibrators substitute for the vocal cords, and between them passes the activating air-stream. The air passing through them was made visible by dropping dry ice into water, which produced a thick, visible vapor. This was then released into a supply tube which led to the model larynx. When this stream as it emerged from the "larynx" was observed stroboscopically, the details of air-flow through the model were revealed. The escaping air was found to be released in puffs, and the pattern of these puffs is described in detail. Between puffs the previously separated "cords" reapproximated momentarily shutting off the air-flow. In order, however, for the series of puffs to be created it is not necessary for the cushions (or "vocal cords") to completely close the glottic chink but, on the other hand, they may remain slightly open.

[*This ties up very well with the opinions of many men whose observations are clinical, as well as experimental*

(Jackson, Negus, Guthrie, Curry, Greene, Russell, etc., all of whom have been cited in this and previous reviews.[2,3] *It is also amazingly consistent with ultra-slow motion photographs of the living human vocal cords, as demonstrated by the Bell Telephone Co. film.* — J. P.]

The author, after a complete study of the pattern of airflow through the model, concluded that in no instance, regardless of variations in the conditions of the experiment, were conditions characterizing vortex tones observed. Eddies were not formed and hence sounds initiated by the cushion-pipe model larynx are not vortex tones. The air stream was rather a series of distinct and separate pulses which themselves functioned as sound initiators. Carhart then postulates, giving his reasons, that the same conclusions apply to the human larynx. Actually, the sound produced is due to the impact of rapidly repeated air-puffs upon the surrounding air, which is comparatively stationary, and which offers resistance to the further escape of the puff itself. The absence of a supraglottic resonator in the model does not affect the conclusions, since a resonator is not the essential part of a vortex tone system. The author concludes that sound production in both the cushion-pipe model and the human larynx is more a puff than an eddy phenomenon.

David Frank,[8] in an article on hoarseness, devotes a few paragraphs to discussions of physiology. He joins the large group who like to compare the physics of sound production at the larynx with that of a reed instrument. He believes it to be the result of vibrations of the vocal cords, set into action by an expiration current of air from the lungs, very much like the vibrations of an elastic tongue in a reed instrument. The cords are first put into a state of tension and the glottic aperture narrowed to afford resistance to the air current. During speech the cords are brought together by the laryngeal sphincteric girdle.

[*This statement must not be misunderstood. The term "sphincteric girdle" is usually used to include the false vocal cords and aryepiglottic folds as well, which do not appreciably approximate in normal phonation.* — J. P.]

Frank continues by telling us that the elasticity of the cords is determined, as Jackson also has pointed out,[1] by the

strength and contraction of the thyroarytenoideii. The elasticity of the glottic margins causes the vocal cords to come into apposition during the lowering of pressure, but immediately as they do so the pressure again rises and the cords are blown apart. This escape of puffs of air is rhythmical and the cords are blown apart.

Aside from this laryngeal mechanism, Frank points out that the lungs, cavities of the pharynx, mouth and nose contribute to normal voice production. The lungs develop the necessary air pressure, and the cavities of the pharynx, mouth and nose modify the intensity of the tone and the relative intensity of overtones.

[*Note that Frank limits the function of the lungs to supplying air-pressure. He does not intimate, as does Jackson,*[1] *that the lower respiratory tract plays a rôle as an air column in affecting the pitch of any given tone.* — J. P.]

James Sonnett Greene,[2] whose many important contributions need no introduction, writes that voice, as distinguished from speech, is simply the production of sound through the medium of expired air. Speech is articulated voice. The normal sounds of speech require not only the vocal cords and pulmonary apparatus but the oral and nasal cavities and the lips, tongue and teeth. Waves set up by the vibrations of the vocal cords are modified by these structures and thus differentiated into the various sounds of speech. The normal and uninterrupted flow of speech depends on proper functioning and co-ordination of all these structures which are under the control of the central nervous system, including association fibres, the speech centre, and centres controlling the peripheral muscles.

The larynx is incorrectly considered as the "organ of voice." In reality it is part of the pulmonary system and its respiratory function is primary.

[*This point is open to considerable argument from the anatomical, evolutionary and physiological standpoints. Effective respiration can be carried out with no physiologically active larynx. A larynx following bilateral laryngofissure is functionless but respiration carries on nevertheless. The same is true of tracheotomized and laryngectomized patients. The*

respiratory function is important but if we must ascribe some "primary" function to the larynx, let it be the sphincteric function which represents the raison d'être of all larynges! Greene, however, I note with much satisfaction, speaks of phonation as "a secondary function." — J. P.]

I have already briefly mentioned Guthrie's article,[5] but have omitted much that it contains. This author reviews many things about which he has written before, and adds further observations. He points out that in an untrained singer the larynx rises as the pitch rises, and vice versa; in the trained singer this movement is reversed and the larynx may actually descend as the pitch rises. The latter movement gives a more pleasing effect at certain pitches due to an increase in the number of overtones. However, the better trained the singer, the less does the entire larynx move.

Guthrie continues by describing his impressions of movements of the laryngeal cartilages. The cricoarytenoid joint is capable of either a rotary or sliding action. Certain muscular actions do not concern the joints at all. The inferior constructor of the pharynx bestriding the wings of the thyroid cartilage may by its contraction narrow the thyroid angle and thus assist to close the glottis. Furthermore, the thyroarytenoid, an integral part of the vocal cord itself, can alter the form, the bulk and the consistency of the vibrating cord and thus alter the pitch. Guthrie thinks this muscle cannot act alone, but is intimately associated with the cricothyroid or external tensor.

The cricothyroid approximates the two cartilages by pulling the cricoid upwards, thus tensing the vocal cords. It also assists to close the glottis, but only with the assistance of the posterior cricoarytenoid and interarytenoid muscles. The glottis can be closed only if a side-slipping of the arytenoids is prevented by the arytenoid muscle, and if the arytenoid is braced back by the posterior cricoarytenoid muscle.

The lateral cricoarytenoid muscle can act either as an adductor or abductor, depending upon which of the other muscles are in action, and upon whether it causes the arytenoid to rotate or slide.

In using the words "tensor" and "tension" as applied to the vocal cords, it must be distinctly understood that no idea of

stretching is implied. The cords vary very slightly in length, if at all, during phonation.

[*In this statement Guthrie is as wrong as wrong can be! The vocal cords absolutely can and do elongate very appreciably during phonation, as the Bell Telephone Co. pictures prove beyond all shadow of a doubt.* — J.P.]

Most important of all is the fact that, while they (the laryngeal muscles) are grouped and paired, each pair does not act as an isolated unit, but always in conjunction with other laryngeal muscles.

Guthrie, in describing the vibratory movements of the vocal cords, says that the laryngeal muscles do not produce the vocal note. Phonation is an aerodynamic, not a muscular phenomenon. The muscles merely adjust and hold the cords in a certain position, at a certain tension and with a certain shape, and the expiratory air current from the lungs does the rest.

We know that vocal cord vibration is mainly transverse, and only to a very slight extent vertical. The larynx can in no wise be likened to a stringed instrument, as alterations of pitch do not depend upon changes in the length of the cords. To some extent the larynx resembles a reed or a siren, but not very accurately. The vocal cords actually are approximated and hardened by muscular action. Then, by increasing pressure of air from the lungs, the glottis is opened and some air escapes. By reason of the elasticity of its margins, the glottis again closes, and by the rapid succession of these movements of opening and closing, a note is produced, the pitch of which depends upon the number of glottic movements a second. The simile of the siren holds good in that the sound is the result of a rapid series of puffs of air. But the number of movements a second depends, in the larynx, upon the rigidity, thickness and tension of the vocal cord, rather than upon air pressure. The mechanism for altering the vibrating mass, such as one finds in the larynx, is not present in any musical instrument. Sir Richard Paget has aptly compared the vocal cords to the lips of a trumpeter.

Laryngeal tones can be produced on inspiration as well as expiration, as occurs in humming, crying, laughing, etc. Even during speech, inspiratory voice is used by children and

very talkative persons. Certain animals, such as the cat and the donkey, can phonate on inspiration.

Experiments, as quoted by Guthrie, of Harless in 1853, and more lately of Ewald and then Negus, with fresh frog muscle stretched across the linear opening of a tube, and stimulated to contract by electric currents and vibrated by air pressure from below, proves that increased contraction and tension raises the pitch of a tone, while loudness is increased by increasing the air pressure.

By stroboscopic studies it becomes evident that vibratory movement in the vertical plane is characteristic of a paralyzed cord, the normal movement being horizontal.

[*I have examined slow motion pictures (128 frames a second) of a patient with left recurrent laryngeal nerve paralysis. Complete compensation has taken place, so that during phonation the healthy cord approximates the paralyzed one. During the production of a tone the paralyzed cord vibrates almost exactly as does the healthy one. The vibratory movements are characteristically wavelike, chiefly horizontal, but with a vertical element as well. During the period of apposition, I doubt that any casual observer could identify the vibrations of the paralyzed cord as being different from those of a normal cord.* — J. P.]

The duration of contact of the cords varies with pitch. Guthrie continues as follows:

In 1832, Lehfeldt suggested that there were two vocal cord mechanisms — the normal and the falsetto — and stated that in the high note of the so-called falsetto register the glottis remained open while the edges of the cords vibrated. In 1932, Husson and Tarneaud, using the stroboscope, observed that in the falsetto register the cords did actually make contact. but that the duration of such contact occupied so small a part of the cycle that it escaped notice under ordinary laryngoscopy. In singing up the scale, the open and closed phases of the vibration vary in duration. In falsetto the closed phase is very brief in comparison with the time occupied by opening and closing of the glottic aperture. It is generally agreed, however, that in the highest notes of the whistle register there does remain a small opening near the anterior part of

the glottis during the entire vibration, and this note is produced only by the rush of air through the hole.

Voorhees[10] cites the correlation between speech defects and loss of hearing by pointing out that the deafened person has difficulty with voice production because he does not know whether he is speaking too loudly or too softly, distinctly or indistinctly. He probably has more trouble with pitch than with quality. Profound deafness in adults brings about a changed quality of speech and intensity. Nerve deafness is always associated with a peculiar voice. Since the patient cannot hear his own voice, he either shouts or mumbles. With conduction deafness, as distinguished from nerve deafness, voices are generally faint, mumbling and indistinct.

Varieties of speech are very numerous and everyone possesses the inherent ability to vary the voice in pitch, quality and intensity. An individual's voice is the result of a settling-down after puberty and is high, medium or low, according to individual characteristics. The spoken voice is, however, a poor index of virility or effeminacy.

Voorhees denies that a person is unaware of the type of voice he has, but knows whether it is high or low, and the volume of the sound produced. The reverse impression is created because people pay only little attention to their method of speech. Voices convey to a listener something by which he judges mentality and strength or weakness of personality. Few succeed in life in mastering men if they have voices that grate on nerves or are otherwise deficient.

Moses,[11] in an article which deals with other things, nevertheless presents a few physiological observations, He says that most vocal disturbances in all stages of life are results of mutation or change of voice. Girls and boys can reveal a prolonged mutation, boys a persistent high pitch — the so-called persistent falsetto voice — without any endocrinologic impairment. Other symptoms of this complex are hoarseness, aphonia and divergence of the chest and head register. Menstruation, pregnancy and senility are likely to cause vocal impairments. Singing during the period of mutation is another problem, since mutation endangers the larynx and voice and constitutes the basis of many vocal diseases. Chil-

dren undergo mutation between 9 and 15 years of age, depending upon race.

Wells,[12] in an interesting article dealing chiefly with hoarseness, presents in the course of his discussion many observations upon laryngeal physiology. He points out that he deals with this phase of the subject in order that there might be a better understanding of the principal portion of his paper. Wells first of all says that the larynx is an organ of "double function," primarily part of the organ of respiration and later developing into the speech organ.

[*This is only partially true, since the author omits probably the most important function of all, namely, its action as a sphincter valve. However, since Wells is simply stating the fact that the phonatory function and the respiratory one are of equal importance, and is not attempting to outline all the functions of the larynx, my criticism is made as a side issue, to stress the importance of the sphincter action in relationship to the other two, and not to create the impression that Wells believes the function of the larynx to be limited to phonation and respiration.* — J. P.]

The vocal cords are spoken of as two "ligamentous bands," although later in the text this author speaks of them as "elastic bands" and points out that they are muscular structures.

[*In a personal communication to me, Wells bases the use of the term "ligamentous bands," to which many will take objection, upon the fact that Spalterholz' standard text on anatomy refers to the true vocal cords as "ligamentum vocale." Wells does not intend to create the impression that the vocal cords are ligaments, but recognizes them as being muscular, and therefore endowed with great elasticity, rather than being of a firm ligamentous nature. This is in keeping with the recognized beliefs of most authorities. The selection of terminology simply creates an erroneous impression not intended by the author.* — J. P.]

Wells points out, as does Jackson,[1] that the action of thyro-arytenoideus internus is responsible for the wonderful flexibility of voice possessed by great singers and adds that the cords can vibrate only when they are more or less in close

apposition, these vibrations causing the stream of air to be chopped into a succession of pulsations which give rise to sound. When the cords are very tense and near together, the vibration frequency rises, producing high-pitched tones; When they are but loosely together the tone is low-pitched.

A whisper is the result when the current of air passes between widely open cords, and is then whipped into speech by the tongue, lips and teeth.

[*The contour of the vocal cords during whispered voice is peculiar in that the anterior segments may be closely approximated, especially in the forced "stage whisper." In this latter case only the posterior segments are widely separated, leaving a large triangular aperture posteriorly, the base of which corresponds to the posterior commissure with the arytenoids separated.* — J. P.]

The whisper contains the essential elements of speech but is lacking in tonal qualities of the vibrating cord. It lacks carrying power, emphasis, inflection and color. With proper vocal action one can speak with little effort, yet be heard, for one thus gets the full benefit of resonance.

Wells intimates that from a consideration of their physical qualities it is probable that the lungs act as absorbers of sound, rather than as resonators. He quotes, in support of this, "some other writer," but without a bibliographic reference, to the effect that "there is no more reason to expect the chest to have resonance than a bellows packed with a damped sponge."

[*This can be successfully refuted by anyone who will consider the effect of percussing a chest. If we tap our finger in thin air a dull, flat note is elicited. If we then tap a finger placed on a chest wall, the sound becomes resonant and louder, which changes represent chest resonance. The difference in percussion note between an area of pleuritic effusion or pneumonic consolidation and over a normal lung area is proof enough of the resonating qualities of the normal chest. It is rather futile to attempt a denial of the fact that the chest is a resonator, and I rather imagine the quotation cited by Wells is from the pen of one totally unfamiliar with physical diagnosis. It is not Wells' intention to wholeheartedly*

support this point of view. He, like all of us, recognizes the factor of chest resonance, but believes it of secondary importance to the supraglottic resonators. In this stand he is supported by certain implications in Frank's paper,[8] already reviewed. — J. P.]

Wells sums up this portion of his article by stating that a good singing or speaking voice depends in the first place upon a good formation and healthy condition of the cords; second, upon a good development of the muscles that activate the cords; third, upon adequate lung power; fourth, upon a favorable development of the resonance cavities of the nose, mouth and throat; and fifth, upon proper methods of articulation.

In his discussion on hoarseness due to laryngeal paralysis, we read that if "there is a complete paralysis of the abductors affecting both cords, instead of hoarseness there will be aphonia, because the cords cannot be approximated in a way to produce vibrations."

[*This statement is due to a misprint. When the power of abduction is lost the cords comes into a position of adduction. In this latter position a comparatively good voice, not aphonia, is present, as can be observed in any case of bilateral recurrent laryngeal paralysis with loss of abductor power. Wells himself, in a recent letter to me, calls attention to this typographical error; he intended to say "paralysis of the adductors," rather than "paralysis of the abductors," and I am privileged to act as his spokesman for the purpose of making this correction.* — J. P.]

The remaining points in this article deal with hoarseness, but since this is a pathological state, a review of this portion of it would have no place in this annual resumé of normal physiology.

Beyer[13] throws a good deal of light upon cricothyroid muscle function in an article dealing with its traumatic paralysis. He points out that there is a great deal of difference of opinion upon the presenting syndrome as a result of this lesion, but expresses the belief that Mygind's conclusions are the most reliable and have been confirmed by experimental work upon animals. The glottis is found to be oblique in unilateral

cases, with the anterior commissure tilted to the paralyzed side. Due to a lack of tension, the paralyzed cord bulges upward on forced expiration and is drawn downward in inspiration, like the flapping of a slack sail. On deep inspiration the vocal cords disappear from view under cover of the ventricular bands.

[*The maintenance of tension must, at least partially, therefore, be a function of the cricothyroideus.* — J. P.]

While Beyer's observations deal with a peripheral lesion of the superior laryngeal nerve supplying a cricothyroideus muscle, Redoglia[29] has had an opportunity to observe and describe changes in vocal cord appearance from a central lesion of the same nerve. By this means he, as well as Beyer, is able to infer a great deal concerning the function of this muscle. Redoglia's case was one of surgical section of the VIIIth cranial nerve, during the course of which operation the IXth, Xth and XIth cranial nerves were injured. Amongst other signs, this led to a complete paralysis of the vocal cord which differed from ordinary recurrent laryngeal nerve paralyses in that the cord at first came to lie in complete abduction. Also, Redoglia states, the loss of function of the superior laryngeal nerve fibres prevented the maintenance of that degree of tension usually found after paralysis resulting from a peripheral lesion of the recurrent laryngeal, in which case the vocal cord is maintained in paramedial position. This central lesion, involving the fibres of both the superior and the inferior laryngeal nerves, lead to a greater degree of flaccidity than lesions of the inferior or recurrent alone, due to the retention of cricothyroid muscle action.

[*It must therefore be inferred, from the articles of Beyer and Redoglia, that the cricoythyroid muscle does play a rôle in the maintenance of vocal cord tension, or else there would be no reason for tension to diminish in the face of its paralysis.* — J. P.]

Lindsay's article[14] on Laryngocele Ventricularis describes this condition fully, but in addition contains many thoughts on the physiology of the larynx in general.

The appendix of the ventricle in the human contains lymphoid tissue and mucous glands which help to supply the

continuous flow of mucous necessary to keep the borders of the vocal cords lubricated during phonation. The larynx can be studied by direct observation, but important data otherwise not determinable can be studied by means of planographic X-rays. Many excellent planographic Roentgenograms are reprinted which convincingly demonstrate the author's observations. These observations deal with the position and shape of both the true and the false cords during the performance of certain functions of the glottis.

During quiet inspiration, with full relaxation, there is a broad aperture between both true and false cords.

[*The width of the glottic aperture varies with the depth and rate of the inspiration. The aperture may not be very great in quiet respiration, whereas in a forced, deep, rapid inspiration the glottic chink may in certain individuals be so wide as to result in the true and false cords lying almost flush against the lateral walls of the larynx.* — J. P.]

During phonation of the letter "E" the true cords are approximated, while the false cords remain apart. Phonation of "E" on inspiration also shows the false cords to be separated.

[*Similar studies upon the production of other letters are unfortunately not described.* — J. P.]

Closure of the glottis on inspiration may show only the true cords to be in apposition, or if the glottis is more firmly closed, both true and false cords come into apposition. This bears out the opinion that the true cords may act as an inlet valve, but indicates also that inlet valvular action of the true cords is supplemented by sphincteric blockage of the upper aperture of the larynx when firm closure is required. Narrowing of the tracheal lumen takes place as a result of the negative pressure.

[*I am at a loss to understand the circumstances under which inspiration takes place with the cords closed. It seems entirely out of reason to visualize the true, or the true and the false, vocal cords as being approximated at the midline during normal inspiration, and even the authors themselves, in a previous paragraph, take the opposite view. We ordinarily visualize them as being abducted to a certain degree,*

sometimes very markedly so, during the inspiratory cycle. In 1938, Canuyt, Gunsett and Greiner,[15] using this same method, presented such observations and pointed out that in inspiration and expiration alike the vocal cords, ventricles and false cords are completely effaced, blending into one another along the lateral walls of the larynx, and thus being abducted instead of adducted.

Looking at Lindasy's planographs, his description seems to be substantiated. I therefore wonder if the author in this paragraph is describing some form of artificial type of inspiration, created for experimental observation, in which the vocal cords are purposely brought together as though for phonation, but instead of producing a note, an inspiration is taken, maintaining the cords in closed position. This seems likely but the point is not made clear in the text. At any rate, the following conclusions are drawn, all of which seem entirely likely. — J. P.]

It is concluded that the true cords may act as an inlet valve, but indicates also that the inlet valvular action of the true vocal cords is supplemented by sphincteric blockage of the upper aperture of the larynx when firm closure is required. Narrowing of the tracheal lumen takes place as a result of the negative pressure. While during simple, normal phonation only the true cords are approximated, in certain pathological conditions the false cords may be used for voice production.

During strenuous muscular activity, such as pulling or pushing with the upper extremities, the glottis is firmly closed by both false and true cords, and the tracheal lumen is increased in diameter, indicating increased intratracheal pressure. The author points out that this finding does not agree with the opinions of Negus. During still more strenuous closure of the glottis which occurs, for example, during straining at stool or during severe coughing, both false and true cords are in apposition, but in some views the ventricle is plainly seen and in some there appears to be more firm apposition of the ventricular bands than of the true cords, as evidenced by the presence of a well-marked notch where the lower margins of the true cords come together. That the intratracheal tension is increased is again indicated by the increased tracheal diameter.

During swallowing the larynx is raised and both the true and false cords brought into firm apposition. Entrance of food into the larynx is prevented by the firm sphincteric closure of the thyroarytenoid, aryepiglottic and thyroepiglottic muscles.

The author draws the following conclusions from his planographic X-ray studies: First, that closure of the glottis during strenuous muscular movements of the upper extremities, during straining at stool or bearing down, and during the early part of coughing before the glottis opens, is brought about partly by closure of the true cords, but to a much greater degree by the sphincteric action of the muscles surrounding the upper structure of the larynx, namely, the thyroarytenoid, thyroepiglottic and aryepiglottic muscles.

Second, that the glottis is closed to prevent the escape of air rather than to prevent inspiration. The intratracheal pressure is increased, and the pressure becomes transmitted to the ventricles, apparently because the true cords afford less resistance than the upper sphincter.

[*This would seem to be excellent proof of a point which I believe is described here for the first time. If the description and illustration of the enlargement of the ventricles does represent an original observation by Lindsay, it is a splendid contribution to our knowledge of laryngeal physiology. The author has furthermore explained the physiological cause for it in a very convincing manner.*

In 1938, A. O. Freedman touched upon the action of the ventricle during closure of the sphincter. He pointed out that in certain species the very large ventricle provided a reservoir for air during the forceful, prolonged use of the forelimbs, which action required sphincter closure and consequent cessation of respiration, but his description does not include the detailed observations of Lindsay as to cause and effect. — J. P.]

Lindsay's third conclusion is that the shape taken by the ventricular bands during apposition, as shown by the planograph, would preclude any involuntary action as an outlet valve. Closure must be maintained by muscular action.

The author points out that almost any situation in which the sphincter comes into play and intratracheal air pressure

is increased tends to distend the laryngeal ventricle, and predispose to the development of laryngocele.

Since Lindsay's article just described has dealt in large part with the laryngeal sphincter, this might be an appropriate moment to review my own article on "The Sphincter Action of the Larynx."[17] Essentially this contribution is nothing more nor less than an outline of the material in Negus' scholarly text, "The Mechanism of the Larynx," and although only one or two bibliographic credits are given to Negus, actually and in fact almost the entire article should be so credited. The chief reasons for having presented this second-hand material at such length are twofold: First, the subject needs to be recalled to mind, since it has been many years since anything in detail has been written upon it and as a result contemporary authors in discussing the broader aspects of laryngeal physiology frequently fail to mention it at all. Secondly, the article acts as a prop for the presentation of original photographs and descriptions of the chain of events as the sphincter opens and closes. Beyond this, it contains nothing that cannot be found in the momentous work by Negus.

Actually, it would be difficult to write upon any phase of laryngeal physiology without simply rehashing material that this brilliant laryngologist has already previously described. The article[17] deals with the evolutionary development of the sphincter of the larynx, its anatomy, muscular control, and mode of action, its nerve supply, function in swallowing, cough, and thoracic fixation, including the rôle of the sphincter in elevating abdominal and intrathoracic pressure. It is then briefly summarized as follows:

The larynx has developed not primarily as a sound-producing organ, but rather as a sphincter valve to isolate the upper from the lower respiratory passages. It first appeared as a circular muscle band in lungfish, to prevent sea water from invading the lungs. There are essentially two laryngeal sphincters, one at the level of and composed chiefly of the aryepiglottic folds, and a second one formed chiefly by the false vocal cords and, to a lesser extent, by the true cords. Closure of this latter sphincter takes place first by a closure of the true vocal cords (which is only partially effective as a sphincter), followed by closure of the false cords.

The purposes of the sphincter mechanism are:

1. To close off the entrance into the lower respiratory tract during swallowing and thereby prevent invasion of this tract by foodstuffs and other foreign matter from above.

2. To permit thoracic fixation by *a.* trapping air within the thorax and *b.* by preventing the entrance of additional quantities from above. This has far-reaching effects in the use of the pectoral girdle and in the establishment of increased intra-abdominal pressure.

3. To permit the momentary accumulation of increased intratracheobronchial pressure before the expulsive effort in cough.

The effect on these functions of the loss of the sphincter apparatus, as after laryngectomy, is briefly discussed.

It is worthy of note that the findings described in this article correspond exactly to those described by Lindsay[14] in his description of the sphincter action as studied by planographic X-rays, and whose article upon this subject I have just reviewed.

By means of a marker connected by a thread to properly curved pieces of brass fitting the thyroid cartilage, Dessy[15] was able to record on a kymograph the movements of the larynx during respiration. Under normal conditions the larynx does not deviate laterally during respiration but, for example, in the presence of pleural effusions, the larynx upon inspiration deviates toward the side of the effusion, or, in bilateral cases, to the side of the greater effusion. This is due to the relative greater respiratory excursion of the unaffected or less affected lung, which causes a deviation of the trachea to the unaffected side, and a corresponding movement of the attached larynx.

By changing the position of the patient in relation to the apparatus, vertical and oblique laryngeal movements as well as lateral deviations can be studied.

Observations made by this method include the following:

There is no difference in the respiratory movement between the sexes. In inspiration, laryngeal movements are downward, often backward, and frequently laterally to the left. The downward movement is brought about not only by action

of the extrinsic muscles of the larynx, but also by the lowering of the cupola of the diaphragm, and the increase of negative pressure within the thorax during this inspiratory phase. These influence the trachea and thus the larynx.

In regard to the A-P movements, in half the cases there is an immobility of the larynx during the two phases of respiration, with the exception of movements due to arterial pulsation. In the majority of subjects an inspiratory lateral movement of small amplitude is present. This is always to the left. Therefore in inspiration, laryngeal movements are downward, often backward, and frequently to the left, but none of these movements are invariably present. The backward and lateral movements occur because the trachea in its downward course proceeds obliquely from front to back, receding from the surface as it goes deeper into the thorax. Therefore the downward movement is necessarily accompanied by a backward movement of the trachea and this is reflected by the larynx. To account for the deviation to the left during inspiration, which occurs more frequently than the backward movement, the author points out that the left bronchus is surrounded in front by the aortic arch. During inspiration the heart is known to descend in the chest, and with it the great vessels, causing traction on the left bronchus, which is, in turn, transmitted to the trachea and thence to the larynx.

Modifications in movements of the larynx due to variations in the type of respiration are reflected almost entirely in the vertical movements. Thus, in an individual breathing abdominally, the downward vertical movements are less well pronounced.

Guthrie[19] substantiates these views, pointing out that inspiration exercises a downward pull upon the trachea and larynx, the normal "tracheal tug."

Stuppler[20] presents interesting information on the nerve supply of the intrinsic muscles of the larynx. He points out that following section of the spinal accessory nerve for torticollis, no change appeared in the action of these laryngeal muscles. Experiments were then carried out on dogs and this finding was corroborated . He concludes therefore, that it is necessary for us to revert to the old theory of Grossman, which held that spinal accessory fibres did not participate in the innervation of the larynx.

[*Certain fibres of the spinal accessory or XIth cranial nerve pass to their peripheral distribution in the trunks of the superior and inferior recurrent laryngeal branches of the vagus. Apparently, however, if these observations of Stuppler are correct, those fibres of the laryngeal nerves which originated in the spinal accessory do not distribute themselves to the laryngeal musculature.* — J. P.]

Canuyt and Gunsett,[21] whose work I reviewed in the April, 1939, issue of THE LARYNGOSCOPE,[2] have repeated many of their previous observations in this current article, and add further new observations. They point out that planographs taken during an apneic period after inspiration demonstrate the vocal cords to approach close to the midline and almost touching. The ventricles are very small and the ventricular bands very large, so that the respiratory slit is very narrow. In apnoea after expiration the image is absolutely analogous. The authors then describe in great detail the appearance of the various laryngeal structures during the production of the different vowels, as follows:

[*I have made these into a table for the sake of simplicity.* — J. P.]

	Ventricles	False Cords	True Cords	Ventricular Slit
a	shallow	not widely separated from true cords	not described	narrow
e	distended a little more than above	separated a very little wider than above	not described	slightly wider than above
i	ventricles distended and puffed up to their maximum	very narrow and extremely distant from each other	narrow as if contracted. Free margins thin and sharp	not described
o	ventricular image smaller. Essentially the same as "e"	see "e"	see "e"	see "e"

[*The above table does not give a complete picture of what happens to each laryngeal structure as the various vowels are produced, but a translation of the text by an expert translator provides no further details.* — J. P.]

In the Valsalva technique of blowing with the nose closed, the pyriform sinus, laryngeal ventricle vestibule and the chink of the glottis are all intensely puffed out. The vocal cords and ventricular bands are widely separated from each other.

In high tones the ventricular bands (false vocal cords) become thickened, larger, more voluminous, and approach each other more than in the normal voice. The true vocal cords are narrower and more flattened. They show a free thinned-out border, and tilt upwards. They likewise more closely approach each other.

[*This observation, that in producing high tones the vocal cords tilt upwards, is of very great importance. It bears strongly upon the question of whether and how the vocal cords undergo elongation and increases of tension as a means of elevating pitch. There is much dissension upon this point. For instance, Dr. Jackson*[1] *cites as a misconception the prevalent idea that the cricothyroideii create tension in the vocal cords, and Guthrie*[5] *believes that the vocal cords do not increase in length as the tension is increased. Some of us*[4,7] *believe that the cricothyroideii, by tilting the anterior portions of the thyroid cartilage upwards, do create increased tension, and, furthermore, that this is accomplished, in contrast to Guthrie's views, by increases in the length of the cords during phonation. Obviously, it is therefore of prime importance for us to actually know whether the anterior portions of the vocal cords can be tilted upwards, since the entire basis of the argument rests upon this point. The findings of Canuyt and Gunsett are therefore significant insomuch as they add an important point in favor of the contention of the group who explain certain elevations of pitch by the action of the cricothyroideii in tilting the thyroid cartilage upwards, thereby elongating the vocal cords and producing a resultant increase in tension.* — J. P.]

Canuyt and Gunsett continue by describing planographic laryngeal appearances in low tones as compared with those of the higher tones, as just described. These low tone appearances consist of the ventricular bands approaching one another, effacement of the ventricles, the assumption of a rounded or globular form by both the true and the false cords.

[*This is another way of describing the generally accepted principle of a thickening of the cords taking place during low tones and a thinning out during the production of higher tones.* — J. P.]
The respiratory chink is smooth and uniform.

In unilateral recurrent laryngeal nerve paralysis, the ventricle is in some cases markedly enlarged and deepened, but this finding may be absent in others. The vocal cord is narrower and likewise the ventricular band. The pyriform sinuses and the external laryngeal recesses are also wider [*due, no doubt, to the flaccidity of the paralyzed muscles surrounding these cavities.* — J. P.]

Calvet[22] reports a case demonstrating the relationship of the type of voice to the state of testicular development. A patient aged 4½ years presented marked precocious hyperdevelopment of primary and secondary sex characteristics. His general appearance was that of a child of 8 years, but the primary and secondary sex characteristics were fully matured. His voice was deep, almost basso. The laryngeal examination presented an adult, completely developed larynx, with projecting epiglottis, a wide-open glottic chink, and thick vocal cords. The voice was able to embrace an octave and the larynx presented the general configuration expected at puberty. The author believed that these changes were due to altered function of the epiphyseal gland.

Motta[23] writes upon this same subject. Following the removal of an adult cock's testicles the cry of the cock remained the same except that pitch was reduced by a half-tone for the first day and a half, the reduced pitch thereafter being less, possibly by a quarter-tone. After bilateral subtotal removal of the testes the range lowered a half-tone, but only for 15 minutes, and then tended to reach normal. After total removal the height of the scale diminished a half-tone, deteriorated in timbre, lost intensity and became hoarse. With one testicle reimplanted into the abdomen, the voice became raucous. There were no modifications of the rhythm. Only the highest four or five notes in the cock's scale were lowered.

[*Apparently therefore no great change takes place in castration after maturity. The findings described represent min-*

imum changes. Possibly there is a change in the quality of the tone, which becomes raucous. — J. P.]

In an article dealing with brain pathology and myoclonias, Kreindler has observed that the laryngeal as well as the pharyngeal muscles might be involved, and undergo rapid rhythmic contractions, unbeknown to the patient, at a rate of 120 vibrations per minute. These contractions are not affected by pulse changes, respiration or sleep. They may at the same time occur in the muscles of the face, thorax and diaphragm. Usually these phenomena are found in the elderly with foci of "brain softening" secondary to vascular changes.

Beau[26] describes in great detail the process of calcification of the various cartilages of the larynx. Since my efforts to abstract the article have not been successful, due to my inability to distinguish the important from the unimportant, I am presenting large portions of it *in toto* so that readers may have this important, detailed study in an English translation. It is as follows:

Calcification of the laryngeal cartilages begins early in life, often between 25 and 30 years of age, or earlier. By 65 years the laryngeal cartilages are almost totally calcified. Most authors do not realize the normal precocious appearance of such cartilaginous changes.

A study of 14 cases of serial ages shows the progress of this calcification. By dying with alizarene, using the gross staining technique of Spalterholz, the bone becomes a cherry red, and cartilage a pale violet, and this method was used in the study. The very earliest stages under 25 years have not been studied.

Ossification begins in the thyroid cartilage and at the same time in the cricoid cartilage; later in the arytenoids, and finally in the tracheal rings.

Thyroid Cartilage: In the thyroid one or several points of calcification appear in the posterior border of the lateral plates. At the same time, or a little later, another nucleus appears in the inferior, middle "tubercle," which soon fuses with the preceding, so that the posterior inferior angle of the thyroid is the first portion to ossify. A second centre appears at the level of the upper part of the angular prominence of

the cartilage just beneath the thyroid incisina, but this centre is not constant and may be absent. At the same age a new point appears at the level of the superior cornu, this point being isolated, but on other occasions this may be due to an extension of the centre located at posterior border. These three primitive points are at peripheral points near the edges of the cartilage.

Originating from these three centres, the ossification at first encroaches upon the borders, and then upon the centre of each lateral plate of the thyroid cartilage. Thus, from the inferior posterior angle two processes spread out. One follows the posterior border of the cartilage, proceeding to meet another process from the superior horn if latter exists, otherwise invading this horn by its own extension. The other follows the inferior border of the cartilage, arriving at the level of the anterior process of the thyroidean angle at the middle level, to meet a similar process coming from the other side. The point situated at the level of the upper cornua, if present, extends a little in the adjacent region. The superior border of the cartilage is the last to ossify. If the angular medial point does not exist, then the process originating from the superior cornua more rapidly invades the lateral plaque and its superior border.

From the middle part of the angular centre there departs very soon a prolongation that invades and fuses with the ossification of the lower border. Moreover, this extends to the neighboring region of the superior border. The ossified zone of the thyroid cartilage then adopts a characteristic form. When the ossification of the borders is completed, then the lateral plaque will also be involved, in part, by extension of the transformed zone of the borders, but especially by the appearance within the medial region of an osseous projection in the form of a tongue. This latter detaches itself from the middle part of the lower border, wandering upwards to join the band of ossification of the upper border. When the transformation is almost completed there remains only two small cartilaginous islands upon each lateral face. These will be invaded much later, and in the elderly the thyroid cartilage is veritably turned into a thyroid bone. There persists at the level of the posterior superior portion of the thyroid plaque a small cartilaginous zone, or even an orifice that seems to serve

as a point of entry. This zone or hiatus corresponds to the course of an arterial branch from the superior laryngeal artery which arises outside the cartilage. It is even possible that the superior laryngeal artery itself perforates the thyroid cartilage at this point.

In women the ossification of the thyroid cartilage is different from that in males. The ossification in general proceeds from the rear to the front by the extension of a band occupying the posterior border and arriving to meet at a medial, inferior angular osseous point.

Cricoid Cartilage: At the level of the cricoid, ossification begins by a small constant nucleus at the posterior face of the angle of the articular surface destined to receive the arytenoid. Toward this same period there appears a point at the level of the frontal angle of this same articular surface, and a third one in the neighborhood of the articular surface for the cornua of the thyroid cartilage. These two latter centres soon fuse and then unite with the first mentioned centres. The ensemble then fuses with that of the opposite side, except a small central cartilaginous bridge that will not be affected except very much later.

Arytenoid Cartilage: The other cartilaginous structures also ossify, and although these have not been studied, yet the following is known: The arytenoids begin to ossify at the level of their base in the region of the muscular process, and in women a little more anteriorly at level of fossa oblongata. They then extend more anteriorly, reaching the vocal process. Then, when the lower half is ossified, a small nucleus appears at the summit (or apex) of the arytenoid.

Cartilage of Santorini: These may ossify, although some authors deny it; some think the cartilages of Santorini fuse with the arytenoids and ossification proceeds from the latter.

Trachea: The rings of the trachea undergo osseous change. It begins at level of second ring by small isolated nuclei which extend circularly along the borders of the ring. The ossification then appears in the lower rings, beginning at the ninth, upon the lateral walls. Later the middle part of the first ring ossifies, after the inferior rings are completely ossified. This ossification may extend until the division into bronchi.

The above, including all cartilages, is only a general scheme, from which there are considerable individual variations, forbidding the formulation of an absolute law. Waldeyer has shown that racial differences exist in the mode and degree of ossification. A knowledge of the X-ray study of these ossifications is now of utmost importance, especially in the study of cancer. Roentgenologically there are discernible three nuclei from which ossification begins:

1. The inferior part of the posterior border of the thyroid.

2. The upper border of the posterior expanded face of the cricoid.

3. At the base of the arytenoid.

These three nuclei are situated very close to each other. From their beginning stage they seem to superimpose or form themselves, one to the other, and resemble the image of an inverted comma sign. The X-ray studies of this ossification are naturally not as precise as the anatomical observations themselves.

Chamberlain and Young[27] have pointed out many cases in which these ossific centres, especially in lateral views, have been taken for foreign bodies, so that their location and appearance must be known for proper diagnoses.

Carcinomata not yet encroaching upon the cartilages may nevertheless decalcify the surrounding areas, in conjunction with a partial calcification of the tumor itself. This is very important diagnostically, so that the normal calcification as it appears on X-ray must be known.

Levin has, as a result of his experiences in the Jackson Clinic, written an excellent article on teaching the laryngectomized patient to talk. Since this article does not primarily deal with laryngeal physiology and yet may be of interest to many, I shall call it to your attention only by presenting the author's summary and conclusions without reviewing it in detail:

"Since the principles of esophageal speech are dependent on the altered anatomic and physiologic status following laryngectomy, the more important characteristic anatomic and physiologic facts are reviewed. The methods of training

laryngectomized patients to talk without the aid of a mechanical larynx are discussed in detail. The surgeon's responsibility should extend beyond the operation and should provide for systematic training for the recovery of speech. Patients can now be reasonably assured before operation that they can learn to talk in a normal way after removal of the larynx. Speech instruction should begin as soon as healing has occurred and the physical condition permits. Esophageal speech is acquired rather easily at this time and the development of speech defects and mannerisms is avoided. It is urged that all laryngectomized patients be first given the opportunity to learn the esophageal method of speech; the artificial larynx should be used only as a last resort for the exceptional patient who cannot master the esophageal method."

I have already very briefly referred to an article by Emil Froeschels.[32] It is, however, of considerable interest and entitled to more than passing mention. The article is concerned with the possibility of the existence of heretofore unknown conscious sensations in the larynx. This was brought to the author's attention in connection with observations on patients with acute paralysis of the recurrent nerve. One group suddenly finding themselves incapable of closing the glottis, instinctively utilized auxiliary muscles of the throat and mouth, producing a "pressure-voice." These patients thereby, without a knowledge of phonetics, make use of a substitute mechanism which phonetically trained physicians attempt to cultivate as a therapeutic measure. The author then asks: "Does not this seem to indicate that something of the physiologic closure of the glottis becomes conscious, even under normal conditions, and that the functions which produce closure do not occur entirely unconsciously?"

[*The answer to this is obvious. It is "Yes." But why Froeschels should refer to these phenomena as "hitherto unknown" is not so obvious. It would seem to me as though laryngeal muscular control has always been considered by all authorities as a voluntary mechanism except for certain respiratory movements which may not be. The vocal cords are controlled by striated muscles, which practically everywhere else in the body are under voluntary control. The nervous controls of the larynx lie in the highest centres of the cerebral cortex. Were the vocal cords not under our conscious*

control, how would it be possible to produce a note of any given pitch? Our speech, if the laryngeal mechanism were involuntary, would result in the production of haphazard pitch which we could not control or vary at will. Everybody, from time immemorial, knows that we can voluntarily control pitch, so I absolutely fail to agree that Froeschels has, as he suggests, contributed anything new. — J. P.]

This author adds that after experimental deafening by a noise apparatus he was able to continue singing a song exactly as before and says that for the first time in his life he "felt" the existence of his vocal cords. His ability to continue the production of proper tones was not due to hearing, but to a sense of "feeling." Experiments of this sort by Froeschels and Weiss have resulted in the observation that "the presence of conscious sensations of movement and position in the larynx has thus been demonstrated" — and that from this fact "the existence of analogous sensations under abnormal anatomic conditions (recurrent laryngeal nerve paralyses) may be inferred." His final conclusion is that "it is significant to be able to demonstrate that there are conscious factors in certain functions which were hitherto considered as belonging entirely to the problematic realm of the unconscious."

[*The author fails to tell us who hitherto considered these functions as lying within the realm of the unconscious, but in a footnote clears up at least one important point to the effect that his own name is sometimes spelled "Fröschels" instead of "Froeschels."* — J. P.]

I have a letter from John C. Snidecor,[33] which, with personal items omitted, I reprint verbatim for the benefit of those who, unlike myself, have a sufficient grasp of the physics of sound to fully appreciate its significance:

"I should like to mention that in loud tones the cord spectrum appears to contain relatively more energy in the higher partials. This would seem logical as the cords certainly would seem to vibrate more complexly under increased pressure. Other research on the acoustic tone as it comes from the mouth would seem to indicate that this energy is passed along and that loud tones as they come from the mouth tend to have

more energy in the higher partials. Our research also tended to indicate that Fletcher's hypothesis regarding the decrement of value in the partials was substantiated in our findings. We found that the intensity of each partial in the cord tone was inversely proportionate to the cube of the number of partials (3.07).

"Regarding the change of voice in the adolescent male, I know that you will find interesting the work done by E. Thayer Curry, now at the University of Oklahoma. He made very exact phonophotographic pitch recordings of preadolescent, adolescent and postadolescent males, and I believe that his research will presently be available in speech monographs published by the *Quarterly Journal of Speech.*

"Some time ago, Dr. Grant Fairbanks at the State University of Iowa became interested in the matter of proper pitch placement for superior speakers with the assumption that the superior speaker uses his voice, by and large, at an optimum pitch level. Wilbur Pronovo, now at Queens College, New York City, has pretty well exploited this subject for male speakers. I later became interested in the problem for superior women speakers and studied the relative pitch level for them. Both Pronovo and I used phonophotographic methods and large samples and believe that we have sufficient data to make some valid assumptions regarding the proper placement of voice levels. This research has only recently been completed and we reported our findings at the National Speech Convention at Washington, D. C., on Dec. 31, 1940. The material is therefore not available in printed form, but the applicable material on placing pitch according to our findings will be found in the section on pitch in Grant Fairbanks' recently published Voice and Articulation Drill Book (Harpers). . . .

"It is my own observation that eunichoid voices in women tend to be lower in pitch than the voices of normal women."

In conclusion, I would like to stress the very great rôle laryngeal motion pictures have recently played in the study of laryngeal physiology and to mention two recent articles dealing with the technique of laryngeal photography, one by Louis Clerf,[34] and the second by myself and Mr. Arthur Hinman.[35]

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PROGRESSIVE DEAFNESS, OTOSCLEROSIS AND CLOSELY RELATED SUBJECTS. AN ABSTRACT OF THE AVAILABLE LITERATURE PUBLISHED DURING THE YEAR 1940.

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(Continued from May issue.)

GROUP 2. MEDICAL THERAPEUSIS AND SYSTEMIC READJUSTMENT.

Crowe,[44] in "Recognition, Prevention and Treatment of Hearing Impairment in Children," did not advocate indiscriminate use of radium as a treatment for deafness. He found that the best results were obtained in children with beginning hearing impairment. Spectacular results in extremely deaf children or adults, were rare. This paper represented a review of the intensive study for 16 years of the causes, pathology, treatment and possible methods for prevention of deafness. Fifteen thousand patients, 3,000 of whom were children, had been examined during this period, by the 1A Western Electric Audiometer, with a range of 32 to 16,384 d.v., tuning fork and voice tests, in a soundproof room.

Most hearing impairment of adult life started in childhood. Therefore, to increase knowledge of causes, treatment and prevention of deafness, concentrated studies on children must be made.

He found that adenoids recurred in more than 75 per cent of the children whose tonsils and adenoids had been removed before the age of puberty. This recurrence interfered with the ventilating function of the Eustachian tube and led to an insidious painless type of progressive deafness. In this type of deafness, hearing was more impaired for high than for low tones. This did not always indicate an inner ear or nerve lesion, even if the hearing by bone conduction was impaired. The examination of children should include a study of the nasopharynx with the nasopharyngoscope; an inspection of

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the tympanic membrane for retraction, especially in Shrapnell's area; a voice test with the opposite ear masked, and an audiometric test with particular attention to 8,000 d.v. and the higher tones. He concluded as follows:

1. Recurrence of adenoids should be regarded as normal.

2. This recurrent lymphoidal tissue may impair the function of the tube and cause low grade tympanic catarrh leading to progressive deafness.

3. Recurred or hyperplastic nodules of adenoid tissue around the pharyngeal orifice of the Eustachian tubes could not be removed surgically.

4. Lymphoidal tissue was so sensitive to radiation that dosage employed was far below the amount causing irritational injury to mucous membranes or surrounding structures.

5. The object of treatment was to reduce size of lymphoid tissue — it had little or no direct action on bacteria.

6. Radiation was of no value in treatment of chronic middle ear suppuration, otosclerosis or any form of inner ear ordinary deafness.

Burnam[45] stated that surgical adenoidectomies in children were insufficient to prevent deafness due to Eustachian tube obstruction. "Irradiation Treatment of Hyperplastic Lymphoidal Tissue" was selected because experiments and observations indicated that lymphocytes and lymphatic tissue was much less tolerant to raying than other nasopharyngeal tissues. The entire rationale of irradiation therapy rested upon such differences in tissues, normal and pathologic.

The most vulnerable part of the cell was the nucleus, and a dividing cell was more sensitive than when in other phases. Reproductive elements in cells were less tolerant than vegetative parts. Nutrition and infection markedly altered sensitivity. To be effective, the irradiation must be absorbed into the tissue.

Two procedures were available. These were the placement of the source of radiation directly in the nasopharynx (radium or its decomposition product, "radon"), or radiation from the surface with X-rays. This author preferred radon or radium

to X-rays. He discussed in detail the technical process of radiation, and described an applicator whose active part was a small glass tube which could be carried back to the nasopharyngeal wall along the floor of the nose.

In this type of treatment, the nasopharynx was considered as two spheroids, and the applicator was placed so that the source of radiation was as near as possible to the center of the spheroid.

Through the walls of the applicator came gamma and beta rays, of which the gamma radiation contained 95.5 per cent of the total radiation, and the beta 4.5 per cent.

The author discussed the Roentgen ray dosage and stated that when both sides of the nasopharynx were treated, 600 to 650 rs. were given. The average dose was 2 gram minutes. Nasopharyngeal inspection and audiometer tests should govern further treatment.

In "Vitamins and the Ear, Nose and Throat," Jones[16] presented a most exhaustive article on the importance of vitamins in the health and disease of the ear, nose and throat. Its purpose was to state, first, the problem of the vitamins and present status; and, second, to present to otolaryngologists a brief resumé of the general literature on vitamins.

According to the author, it was well established that certain vitamins have a special effect upon certain structures, and he had analyzed the structures in the ear, nose and throat derived from the three embryonic germ layers. In the internal ear all epithelial structures in cochlear and vestibular portions, as well as nervous tissues, were derived from the ectoderm. The supporting structures of the membranous labyrinth and the bony otic capsule came from the mesoderm. In the middle ear the epithelium was derived from the endoderm, and the ossicular bone from the mesoderm. In the tympanum, the bone, the muscles and supporting tissues were from mesoderm, the lining epithelium from endoderm. In the Eustachian tube, the cartilage and bone were from mesoderm, the epithelial lining from endoderm.

Each vitamin seemed to have a preponderant effect upon special structures. Vitamin A appeared to be particularly concerned with disease and health of the epithelial tissues,

derived from both ectoderm and endoderm. Vitamin B was actually the anti-neuritic vitamin and had a particular relation to nervous tissues derived from ectoderm. Vitamin C was apparently directly related to tissues derived from the mesoderm, connective tissue and blood capillaries; and vitamin D unquestionably related to bony structures.

The author stated that no vitamin could be expected to improve any condition not caused by lack of the same vitamin, and that the problem was not one of advanced vitamin deficiency in which the diagnosis was simple, but in the great number who had only a mild deficiency. Certain laboratory tests were available; *e.g.*, the amount of pyruvic acid in the blood and urine. If above the normal level, vitamin B therapy was indicated. Ascorbic acid could be detected in the urine and blood plasma and, if below normal, vitamin C was indicated.

The author stated, in reference to improvement of function in the internal ear, it was necessary to postulate the following:

1. Impaired function must be directly due to lack of vitamins.

2. The cells of Scarpa's ganglia and of the spiral ganglia must still be viable.

He pointed out the many natural sources of vitamins and emphasized the fact that a vitamin was not a drug. He stated that at the present time the only way we could regulate dosage was to prescribe the vitamins themselves, and we should supplement all vitamin therapy with foods which were high in vitamin content. Drugs such as iron, thyroid, alcohol, and physical exercise, all complicated the dosage.

In this article the author reviewed the clinical studies of Selfridge and the histopathology of Covell in their work with vitamins in connection with the ear, nose and throat.

According to Childrey,[47] in "Vitamin B and Other Measures in the Treatment of Deafness," but little moral support could be offered to patients with otosclerosis, perceptive deafness from trauma, neurotrophic infections, senile deafness and many middle ear deafnesses of long duration, even without affecting perceptive elements.

It has been estimated that 10 per cent of the population had defective hearing.

He stated that with further appreciation of the limitations of hearing aids and therapeutics in deafness, the physician would be enabled to devote more time to preventive phases, which should include routine examinations and, if necessary, training in lip-reading. Hearing aids were unsatisfactory when there was much impairment of the perceptive element, and to obtain the greatest benefit, required patience and re-education of the patient. The electric hearing aids amplified all sounds without selection, and in patients with nerve deafness this amplification might include head noises and tinnitus and produce fatigue in hearing, and possibly some auditory nerve damage. The patients with perceptive deafness heard best in the morning.

When a cause was discovered, as a deafness from specific disease, allergy, drugs, impacted cerumen, or exposure to loud noise, partial or complete recovery might occur if the hearing was not impaired too long. Most nonspecific nerve deafness occurred in elderly people and the hearing normally decreased with each decade after the fourth. Normal hearing occurred in about 16 per cent of persons over 65 years of age.

In reviewing some surgical procedures for deafness, he remarked that, according to Holmgren, suitability for the fenestration operation required an infection-free tympanum, an ankylosed stapes and an appreciable perceptive element remaining. The intranasal operation for securing a permanent window of the antrum was brought into the comparative analysis.

In stressing the importance of preventive measures, he pointed out that the important ones were avoidance of loud noises, control of the hereditary factor in otosclerosis, adjustment of nerve pressure in high tensioned individuals, the early treatment of the nasopharynx and sinuses for catarrhal deafness, avoidance of nose blowing and swimming, the use of vitamin B, especially the thiamin chloride element, and the restriction of tobacco, alcohol, certain drugs and fluids, and discussed the possible value of the Furstenberg salt-free regimé.

Successful results in the use of synthetic vitamin B were discussed by Brandenburg[48] in "Relief of Neuritis of VIIIth Cranial Nerve with Vitamin B." After reviewing the work of other authors, he carefully reported one case and concluded that parenteral administration was desirable for producing rapid saturation, because a neuropathy of the vagus nerve might have resulted in inactivity and even atrophy of the glands of the gastric mucosa, interfering with normal digestion and absorption from the gastrointestinal tract.

Thiamin chloride could be given subcutaneously, intramuscularly, intravenously or even intrathecally with a wide margin of safety, as 25,000 to 50,000 times the average daily requirement was necessary to produce lethal effects.

To prevent recurrence of symptoms, 50 international units were necessary for children, and 300 for adults. One milligram of thiamin chloride was equivalent to 330 international units.

In his summary, the author reported one case of severe bilateral tinnitus and impairment of hearing following extensive radiation of the cervix for carcinoma, which was relieved by the daily intravenous injection of thiamin chloride, from 10 to 15 mg. doses, for nine days. He further suggested the advisability of intravenous administration of thiamin chloride for all neuropathies of the cranial nerves.

In "Nature of Vitamin B and Its Components, with Special Reference to Nerve Deafness," Veasey[49] has attempted to clarify the relation of the deficiency in vitamin B to certain forms of nerve deafness, discussed the chemical and biological nature of the catalyst, and reviewed the literature. He commenced the latter with historic allusion to the study of beriberi in China as early as 2600 B.C. and discussed its relation to toxicity or deficiency in polished rice.

The six divisions of the B group were outlined, but special importance was given to thiamin (B_1), riboflavin (B_2G), nicotinic acid and the factor of gyorgy (B_6).

In summarizing, the author stated that a vitamin B deficient person might present symptoms referable to the nervous, vascular or gastrointestinal system, endocrine glands or the organs of special sense; even without pathognomonic

lesion or syndrome, he would be below par and in the neurasthenic list.

This summary did not adequately represent the physical and chemical reactions brought out in the studies of these vitamins. Deafness was rarely mentioned in the various references but the author maintained that it should be included as a possible clinical manifestation of vitamin B deficiency.

The laboratory studies of vitamin B reaction, standards of measurement and requirements in man were extensively reviewed. This review included both quantitative index, chemical estimate and biologic studies (deficient rats with neuritis).

Cowgill had realized that the thiamin requirement was related to caloric intake and body weight, and by formula evolved the rule that for a man weighing 70 kilograms, on a 3,000 calorie diet, the adult daily thiamin requirement would be 300 international units. The conditions which were known to modify the requirements of vitamin B_1 were stated as follows by the author.

"It is increased by a high intake of carbohydrate and is spared by high ingestion of fat. It varies with the species and with the individual. It is greater in the presence of infection, fever, diabetes, hyperthyroidism, pregnancy, exercise, a high basal metabolic rate, lactation, an unusual gain in weight, elderliness in woman, a postoperative state and any gastrointestinal condition causing poor absorption, such as colitis, diarrhea, pyloric stenosis or achlorhydria."

The writer has presented careful tables for the balancing of vitamin B deficiency in dietary intake, discussed the effect of cooking, reported seven cases of his own inconclusive experience in vitamin treatment for impaired hearing. In his conclusion, he presented clinical difficulties in meeting vitamin B deficiency, the inaccuracies in food content, and selection of patients.

Voorhees[50] presented a study on "Defects in Speech in Relation to Defects in Hearing." He contended that speech defects were even more common than defects in hearing, and the profoundly deafened person had particular difficulty with

the loud and soft tones of voice production. He discussed the subject under five headings:

1. Anatomic defects, often congenital; *e.g.*, cleft palate, harelip and dental malocclusion.

2. Severe colds in childhood superinduced by excess lymphoidal tissue in the nasopharyngeal area.

3. Speech defects in certain frequencies, usually associated with defects in articulation.

4. Profound congenital or acquired deafness in children. If deafness occurred after speech development, there were less marked vocal defects, though often a muffled or denasalized tone.

5. Profound adult deafness with changed quality and intensity in speech.

The author emphasized the importance of the regulation of speech training.

In "Otosclerosis Treated with Sex Hormone," Bernstein and Gillis[51] pointed out that otosclerosis was one of the most common and most serious causes of progressive deafness. Because it was familial and hereditary, was much more common in females, often began at puberty, tended to stop at the menopause and was made worse by each pregnancy, they thought it might have some relation to the sex hormones. In a normal pregnancy the amount of estrin in the blood was greatly increased, and in a susceptible patient the otosclerosis increased. Administration of sex hormones caused dilatation of the peripheral vessels and increased circulation of the blood. This was more manifest if the heterogenic hormone was given. Increased peripheral circulation might cause absorption of the embryonic bone in otosclerosis.

They treated 56 patients. Eighteen males received injection; seven were improved and 11 were not improved. Thirty-eight females were treated; 24 were improved, 14 unimproved. The authors gave several preparations:

Perandren	5-10 mg. weekly
Stilbestral	1 mg. daily
Ovocylin P.	5 mg. weekly
Progynon	10,000 units weekly
Estrone	20,000 units weekly

When one or two of the preparations failed to give desirable results, the other preparations were used. The patient's hearing was tested by whispered and spoken voice. The authors felt that sufficiently good results were obtained to encourage further investigation and that there was now much more hope for the otosclerotic patient.

That more than 50 per cent of the deafness which developed before puberty might be prevented from reaching a very severe degree or may be prevented altogether, was stated by Bordley[52] in "The Treatment and Prevention of Deafness in Children." The difficulty lay in early recognition of hearing impairment in children and in the early discovery of the predisposing factors in deafness.

The author emphasized the fact that to prevent deafness, children of early school age should have very careful examination of their hearing, as well as otoscopic, nasopharyngoscopic and audiometric examinations. The high tones especially should be tested. The immediate eradication of infected lymphoid tissue should be undertaken whenever it was found involving the Eustachian tubes. Allergic manifestations should be corrected.

Careful treatment of acute infections of the middle ear and mastoid with such drugs as sulfapyridine and sulfanilamide, combined with conservative surgical measures, offered untold possibilities for the prevention of deafness in later life. All children with impairment of hearing should have periodic check-ups.

Prietzel,[53] in "Generative Gland Treatment in Cases of Senile Deafness," stated that various methods had been employed in different fields of medicine to conduct intense research with the use of generative gland preparations. Clinically, senile deafness presented the picture of normal tympanic membrane, lowered upper tone limit, diminished acuity for higher tones and shortened bone conduction. Tinnitus aurium was usually present. The pathology underlying this condition was a change in the basal membrane of the cochlea with the greatest involvement in the vestibular blind sac.

In this research, men and women between the ages of 50 and 70 years were studied. They were all treated with male

sex hormone preparations. There were no counteractions between male and female sex hormones.

Loeser stated that testosteron did not have a direct effect on the sex organs but on the anterior pituitary, perhaps the negative adrenotrope, and the effect of reduction of blood pressure appeared to have very real significance in the treatment of senile deafness.

In senile deafness, tinnitus aurium was the most prominent and frequently occurring symptom. Injection treatments always resulted in great improvement in hearing; *i.e.*, the tinnitus aurium symptom disappeared. High blood pressure was reduced after treatment. "Perandren" and "testoviron" were the preparations used and seemed equally efficacious. The selection of cases was important. Intramuscular injections of 25 mg. were given twice weekly. Koch had demonstrated the absolute failure of the injection method in cases of otosclerosis.

The author concluded that the ideal cases for this type of treatment were those with tinnitus aurium as the predominant symptom and those with this symptom for only a short time.

Ponsan[54] discussed "An Interesting Method of Treating Deafness" and described the use of the Itard sound which he introduced into the orifice of the Eustachian tube, and then blew sulphur vapor into the middle ear cavity with a rubber bulb. The sulphur covered the mucosa of the Eustachian tube and middle ear, and in certain cases gave excellent results. He felt that the cicatrizing and decongestive action of sulphur vapor was certain in catarrh of short duration but was of no value in progressive deafness of long standing.

The new method he suggested was a combination of intermittent pneumatic massage of the tympanic membrane and sulphur insufflation.

His procedure was to use a Siegel speculum, which kept the ossicles constantly in motion and at the same time insufflated the sulphur vapor. The main point was to adapt to the individual case the pressure and duration of insufflation and massage. He stated that in his opinion tubal catarrh was caused by too great dilatation of the Eustachian tube, and

this constant communication between the pharynx and middle ear caused arthritis of the ossicles, and he believed that in the pathology, as well as the therapy of this condition, the old conceptions must be abandoned.

The indications for this treatment were catarrhal otitis which responded best; suppurative otitis media which responded favorably, and mucopurulent otorrhea, particularly with cicatricial or adherent otitis, which responded satisfactorily. He reported five cases showing marked improvement from this treatment.

In "Vitamin Treatment of Otosclerosis," Baer[55] stated that constitutional factors played a rôle in the otosclerotic process of new bone formation, that the therapy of this disease was still an obscure problem, and that the methods used had been almost entirely unsuccessful. Mechanical treatment in the form of radium, X-rays, diathermy and surgery had been successful in some cases. He suggested the probable connection between otosclerosis and vitamin deficiency, and commented on the osseous changes which occurred in both diseases. He had found vitamin B to be effective in the treatment of tinnitus aurium; vertigo and other manifestations were definitely associated.

The author reported that his experiments on 15 patients with otosclerosis treated with a combination of vitamins B and C gave negative results, but that he was still hopeful that the vitamin deficiency problem might solve the difficult question of therapy of otosclerosis.

Selfridge[56] presented a contribution on "Chronic Progressive Deafness with Special Reference to Estrogenic Substances," particularly the ovarian hormone and its importance to both men and women. After presentation of important views from various authorities, in conclusion he made valuable comments from combined opinions.

The author quoted Gray in the sex percentage ratio for otosclerosis as 40 per cent for men and 60 per cent for women, and the latter's belief that the essential causative factor in otosclerosis was a gradually increasing defect in the vasomotor mechanism which governed the nutrition of the organ of hearing as a whole. He cited Langdon Browne's opinion that the male and female hormone were closely related

chemically, and McLellan's belief that boys who responded to estrogenic treatment might have been subjected to an excessive amount of estrin or to a deficient amount of anterior pituitary substance during intrauterine life. Emphasis had been placed upon vasomotor disturbance at the menstrual and menopausal periods.

In a recent group of cases studied by the author, a scanty menstrual period, marked nervous and emotional episodes, indicated estrogenic need; as many individuals showed a lowered basal metabolism, the use of estrogenic substances before the addition of thyroid often brought about a definite improvement in impaired menstrual functions, nervousness, fears, fatigue, tinnitus, and some improvement in the hearing curve.

The author quoted freely the work of Hayden on lowered basal metabolism; Mortimer, Collip and Wright on both atrophic rhinitis and otosclerosis, and various authors on the variations in calcium deposit. Besides estrogenic, the thyroid, pituitary and adrenal cortex substances were important in treatment. Among vitamin factors, thiamin chloride and nicotinic acid were emphasized.

Clinical histories were presented.

Nerve and conduction deafness appeared to be linked to many factors involved in growth, such as endocrine glands, vitamins, mineral salts (the electrolytes) and amino acids.

Nutritional deficiencies during the period of gestation indicated that the prevention of deafness depended upon an optimum diet for the mother.

It was suggested that endocrine and allergic factors might prove related to tonsil and adenoid hypertrophy.

Hays[57] philosophized about the 5 per cent of the population who have hearing defects, in "How We Can Overcome the Handicap of Deafness," and wondered whether there would be less deafness in the future since routine audiometer tests were made in the schools. He also speculated as to whether the untold thousands would take advantage of lip-reading lessons or wear a hearing aid. In an analysis of 2,019 cases of impaired hearing in New York, the cause of deafness was

found to be: diphtheria, 7; scarlet fever, 27; measles, 212; mumps, 15; chicken pox, 25; rheumatism, 3; pneumonia, 18; meningitis, 9; accidents, 26; pertussis, 27; influenza, 6; colds, 46; congenital, 13; tonsillectomy, 2; infantile paralysis, 1,335; and exanthema, 237.

The author stated that in overcoming the handicap of deafness there were two problems:

1. The prevention of deafness and the medical treatment of the ears themselves.

2. Adjuvant to treatment, study along educational lines of lip-reading or the use of an electrical hearing aid.

The prevention of deafness was our most important problem. Retardation and lowered intelligence were not the only handicaps accompanying hearing defects. Emotional and personality difficulties appeared with or subsequent to hearing deficiencies. When one teacher in a school read the list of hard-of-hearing pupils in her school, she exclaimed: "You might as well have given me a list of my problem children."

If one desired to overcome the handicap of deafness, he must start when the deafness was in the incipient stage. Improvement could be obtained by building up the general physical condition of the child, attending to local causative factors in the nose, throat and ears, and treating the ears themselves. If the hearing could be improved in the early years, there was possibility that improvement would be permanent. The author stressed the importance of early instruction in lip-reading for children whose deafness did not respond to treatment.

Nodine[55] discussed "The Hard-of-Hearing Patient; Recent Advances in Treatment." He cited the work of Jarvis and stated that under proper dietary regulation individuals without suppurative processes in their ears had gained from 10 to 40 per cent of their lost hearing. The diet consisted of foods low in carbohydrate content and high in citrus fruits, supplemented by mixed vitamins in capsule form, and in children some form of iodized cod liver oil. The use of estrogenic sprays for reduction of tinnitus was mentioned, as well as surgery of tonsils and adenoid, and deflected septa.

He stated that the most useful and practical treatment in the field of restoration of hearing, was the long-continued use of 50 mg. of nicotinic acid, t.i.d., and commented on the use of sulfanilamide in otitis media and the lavage of suppurating antra with lipiodol in order to stop the infection and its influence on the ears.

He reviewed the surgery of fistulization in otosclerosis and tabulated Lempert's indications for the operation.

Under Clinical Notes, Roth[59] described "A Tuning Fork Audiometer" with a series of calibrated forms. A cup-shaped collector of sound at the end of the stem, and a cross pin for sustaining the fork between thumb and finger were new features. In his comments, the author claimed that the forks, after calibration, could be used as a standard for calibrating electrical audiometers, and the electric audiometer for calibrating the forks. The forks remained constant in pitch and intensity for potentials with no wearable nor varying elements, such as occurred in intricate electrical hook-ups. There was no bone conduction factor of error when the sound collector was in contact with the auricle.

In "Audiometric Range in Allergy," Greenwood[60] pointed out that up to date allergic states of the middle ear which clinically appeared to be present, were not demonstrated by cytologic and pathologic investigation.

In view of the continuity of mucous membrane lining the tympanic cavity with the Eustachian tube and nasopharynx, and the nasal mucosa response to allergens, studies were made to determine whether there might not be an impairment of hearing simulating a conductive type, from the waterlogging of the mucosa of the middle ear. Sixty-five unselected allergic individuals were studied. In this group, six had impaired hearing, with one definitely worse during allergic attacks; four had tinnitus, and five had vertigo. The tympanic membranes showed changes in only thirteen cases, ranging from dusky hyperemia to a thickened opaque membrane.

The allergic cases showed marked drop in the lower tone limit.

As only audiometric records without cytologic sections were reported, the conclusions were apparently incomplete.

In the field of "The Genito-Nasal and Genito-Aural Relationships," Mortimer[61] first reported in 1936 on the intranasal administration of estrin in atrophic rhinitis, and stated that six females and one male were found suffering from progressive deafness, as well as atrophic rhinitis. More recently, the author and his associates reported on a group of 250 patients, in which 42 were found with both progressive deafness and atrophic rhinitis. In a group of 35 cases of constitutional deafness, the two defects coexisted in the same individual, and in the same familial stock.

They found that the treatment which acted specifically upon the nasal disease, not only was capable of producing marked improvement in certain of the aural defects but there was improvement in the hearing level of the constitutionally deaf as a whole.

Although but a preliminary report, the author believed there was definite evidence of the lowering of the threshold of hearing, a change in acuity and evidence of genitoaural relationship in man.

"Effect of Impaired Hearing on Youth" was discussed in a general way by Hays[62] on the social and economic sides. A group test of 792,913 children during four years showed a hearing impairment in both ears of 37,798. The author concluded that 5 per cent of school children, many in the youth group, had impaired hearing in both ears.

He urged suitable treatment for curable cases of deafness, and in the last analysis advocated lip-reading and adequate earphones in the readjustment problem.

That the problem of the hard-of-hearing was no longer an individual one but a public health problem, and, as such must be met by the community in order to cope adequately with the situations involved, was the opinion of Zwerling[63] in "Hearing Impairment; A Public Health Problem."

There were approximately 10,000,000 adults and 1,000,000 children with acquired hearing impairment. The program set up involved the early discovery of hearing impairment, parental co-operation, educational and vocational guidance (bookkeeping, designing, art, dressmaking, filing), and remedial treatment, in so far as that promised results. The social

problem entered in the deafened person's relationship to family, friends, neighbors, teachers and employers. The author made a plea for further work by public health-minded and public-spirited individuals, such as the American Society for the Hard-of-Hearing and the New York League for the Hard-of Hearing, who have done splendid work in meeting the problems of the deaf, conducting research and maintaining clinics.

After reviewing the general commercial side of hearing aids, in "Prescribing a Hearing Aid," Day[44] stated, "Lack of acceptance and adjustment to their handicap has kept many of the hard-of-hearing public from using hearing aids." This was increased by a certain amount of discomfort in the wearing, amplification of the spoken word, and many annoying and adventitious sounds. Only when they had emerged from this depression, inferiority and self-pity, and patients were ready to help themselves and receive help, could the hard-of-hearing profitably utilize a hearing aid.

The duty of the otologist to the hard-of-hearing patients, did not end with finding that physical remedies were unavailing; with tuning forks, audiometers to determine the type and degree of deafness, he should advise the most suitable aid and discuss their problems with them. The author stated that there were two general types of electrical hearing aids on the market: *1.* the carbon or telephone type of aid, with microphone, carbon amplifier and earpiece; *2.* the vacuum tube or radio type. Carbon aids were light and portable; though the cost was high, it was less than for the vacuum type, and the upkeep was moderate, batteries lasted longer and were readily concealed.

In the range of amplification they rarely increased tones below 500 or above 2,500 cycles. Although the range of speech is usually between these points, the overtones up to 5,000 cycles gave quality to the voice. The amplification was, however, not uniform, and selective amplification was impossible with the present carbon hearing aids; on the other hand, the vacuum tube type of hearing aid gave undistorted amplification throughout speech and musical range. A small portable type was now made, apparently giving faithful amplification. The vacuum tubes were more expensive and broke easily.

Transmission of sound by either air or bone conduction could be supplied by both types. When deafness was primarily conductive and there was good hearing in the higher frequencies, bone conduction was preferable. The air conduction instrument was better for perceptive and nerve deafness. In mixed deafness, complete air conduction and bone conduction audiometer study was necessary to determine which type of receiver was most efficient. In conclusion, the author stated that average hearing loss must be 25 to 30 dcb. before a patient would tolerate a hearing aid for general use.

The carbon type of hearing aid was at present preferable to vacuum type in conductive or mixed deafness where there was relatively good hearing above 2,000 cycles. In cases of average hearing loss greater than 50 dcb., a high quality vacuum type was preferable.

According to Wells[65] in "Some Practical Considerations with Regard to Hearing Tests," principal deafness etiology was: *1.* cold infection; *2.* toxins within and without the body; *3.* excessive noise, particularly traffic and transportation.

The author believed that with properly conducted hearing tests in soundproof room, at least 10 per cent of the populaton, 13,000,000 people, would show at least 10 per cent loss of hearing. According to the U. S. Bureau of Education, 500,000 children needed special educational provision for defective hearing.

The purposes of the hearing test were: *1.* differential diagnosis in impaired hearing; *2.* determination of need and type of hearing aid; *3.* evaluation of school ability and classification of school children; *4.* examination of preschool children for similar reasons; *5.* determination of vocational fitness; *6.* determination of fitness for military duty; *7.* determination of value of methods of treatment; *8.* social and marriage considerations.

Hearing tests were both to determine capacity of hearing and the underlying cause for deafness. The former could be undertaken by laymen; the second should be by specialists.

The audiometric instrument with phonograph attachment was valuable in the examination of school children, but more

accurate tests for stenographers, musicians, aviators and military people were necessary.

In summary, the author stated that hearing tests were obviously the province of the ear specialist, though simple acuity reaction could be made by laymen. The author suggested the value of an electrically-operated unitone instrument of watch tick type.

In "Hear and Now," Hayden[66] stated that people with hearing losses were of two types: the "deaf" and the "deafened." The truly deaf were a small group of persons who were born without hearing organs, or whose auditory powers were destroyed before they learned to talk. These persons must be educated in special schools and their problems were comparable to the problems of the blind. In sharp contrast were the millions of deafened persons with varying degrees and types of impaired hearing, who formed an exact parallel to those who must wear spectacles to compensate for some defect in vision. Practically all of these handicapped persons could definitely be helped by recent advances made in diagnosis and treatment of deafness.

In discussion of the mechanism of hearing and the way disorders so frequently occurred in the middle ear, the author pointed out that every time we breathed, a fresh supply of air was drawn into the cavity of the middle ear through the Eustachian tube. This was a highway for bacteria immigrating from the nose and throat. Swimming and diving in even pure water opened avenues of infection that facilitated middle ear infections. Poor food often meant poor hearing. Tests on 100,000 English school children demonstrated that middle ear deafness was four times more prevalent under poor social than under good conditions.

The author stated that the main cause of middle ear deafness, suffered by several million people (mostly beyond the age of 40 years) was otosclerosis.

Comparatively rare was deafness caused by damage to the inner ear. Inadequately treated syphilis and cerebrospinal meningitis could impair the auditory nerve; so could tobacco and alcohol poisoning. Lip-reading was the best method of salvation in this group.

The other group, instead of having psychologic depression, gloom and oversensitiveness, could be helped now with electric hearing devices. These were of two types, the air conduction and the bone conduction.

Another advance in hearing aids was made two years ago with the introduction of vacuum tubes identical with radio tubes. These were made to compare in size, weight and cost with the best carbon sets.

The prescribing of hearing aids was now out of the "hit and miss" class by the use of the audiometer. The principal objection voiced by the patient against hearing aids was "I'm not deaf enough to need a hearing aid." Others felt that it would affect their hearing adversely. "It costs too much" was a common objection. The main reason was a deep-rooted prejudice which was inexplicable.

Many cases of deafness could be prevented by removing diseased tonsils and adenoids, proper care of ears during the acute infectious diseases, and periodic examination of children's ears.

Behnke[67] wrote on his experiences in the Navy in "Noise in Relation to Hearing and Efficiency" as a preventive problem for Naval Medicine.

He stated that noise was an unpleasant or disturbing sound and that it usually contained a large number of frequencies. He stressed the fact that the sensation of loudness, as recorded by the ear, did not exactly correspond to the intensity scale as measured in decibels except for a comparatively small band in the frequency range. No comparisons and no valid record of noise levels could be made on the basis of subject reactions. The use of sound measuring instruments alone or in combination with the ear was imperative in the study of the noise problem. Numerical values on the decibel scale did not correspond to differences in sensation. If the intensity of a sound source was increased to 30 dcb. above its original level, the average person would consider the sound level to have increased tenfold. It was the individual's emotional reaction that rendered sensory perception with respect to hearing so unreliable. Noise audiograms taken in New York showed a deafening effect of 45 to 55 dcb. in offices and places of busi-

ness, while on a noisy street corner, sounds of less than 80 dcb. would be masked.

In a group of 1,040 workers, the greatest incidence of deafness occurred in those groups subjected to the greatest amount and to certain types of noise. Fifty-two percent of train dispatchers suffered from diminished hearing in the "telephone" ear. Particularly damaging were very loud or sudden noises, such as explosions. Gunfire deafness was a particular concern of the military service. The factor of bone conduction vibrators in the etiology of noise deafness appeared to be important, as judged by results of animal tests. Efficiency had been found to be increased from 5 to 10 per cent with elimination of noise and by the use of ear protectors. Cotton ear plugs reduced noise intensity by about 10 dcb. Specially devised protectors reduced air-borne sound from 25 to 30 dcb.

Rebattu[65] reviewed the history of the "Otologist and Deafness" and gave some consideration to the mental effects produced by deafness, and mentioned famous persons so afflicted.

He stated that Guyot, a layman, who was deaf, first had the idea of treating deafness by way of the Eustachian tube and from that time the attention of otologists had been concentrated almost exclusively on catheterization. Saissy proposed lavage of the middle ear through the tube, and Itard used medicated vapors. After the discovery of electricity, great hopes were based on the use of galvanism. Raviremus proposed perforation of the drumhead to permit air to enter the inner ear.

Jasser carried out mastoid trepanning suggested by Riolan. Surgical treatment of deafness was discussed at the International Congress in Rome in 1894 and at the Congress in Paris in 1900. The conclusions were not encouraging. According to Siebenmann, effective therapy would mean a re-establishment of mobility of the stapes or a replacing of the stapes by a mobile cicatricial membrane.

Bárány (1910) found that a previously constructed labyrinth window could be opened into the semicircular canal, either at the level of the oval window or the promontory.

Wittmaack and Holmgren described operative techniques to reach the semicircular canals in such a way as to avoid infection of the labyrinth and suppuration of the middle ear.

Ten years ago, Sourdille studied the surgical treatment of deafness and developed a method which consisted of a tympanolabyrinthopexy with a view to re-establishing tympanic function inhibited by ankylosis of the stapes. He stated that results had been encouraging, but that the method was suitable only for specially trained surgeons.

In his President's Address, Newhart[69] made "Observations on the Conservation of Hearing," and in explanation of the retarded progress in prevention and alleviation of conditions causing hearing difficulties, cited, first, the anatomic inaccessibility of the ear for research, diagnosis and treatment; second, the inadequacy of the classic hearing tests for the accurate detection and measurement of hearing deficiency; third, the persistence in belief that efforts to prevent, cure or compensate hearing defects were most generally futile; and, fourth, the lack of familiarity among physicians, educators and public of the fundamental facts, one of the most important of which was that many individuals had unrecognized handicapping hearing impairment.

In solution of this problem, a notable impetus to progress had occurred through the employment of the audiometer in research, clinical practice and early detection of hearing loss.

First in importance was the periodic testing of school children; in many States this was required by law. In 1938-1939, 1,900,000 pupils in the United States were given audiometer tests. The Western Electric phonograph audiometer, testing up to 40 pupils at a time, was generally used. There had been a lack of medical follow-up in this work. The limitations of the test were, first, this type of instrument could not be used advantageously for the rapid, simultaneous testing of large groups of children too young to write dictated numbers; second, the phonograph audiometer failed to disclose hearing impairment for the higher frequencies.

A simple pure tone audiometer had been designed and made available at low cost for expeditiously screening larger groups of school children.

In tests reported from the Minneapolis public schools, out of 6,344 tested, 1,260, or 19.8 per cent, deficiencies were found. Of these, 14.4 per cent had losses only for the two

higher frequencies, 4,096 and 8,192, and 344, or 5.4 per cent, showed loss only for frequencies below 4,096.

These preliminary findings suggested the importance of having one tone frequency above 8,192 cycles, such as 11,548 cycles, included in the school tests for screening purposes. The following important steps should be seriously considered:

The early fundamental teaching to undergraduates of: *a.* the technique for the discovery of hearing defects; *b.* the prevention, correction and amelioration of hearing handicaps by modern methods; *c.* the importance of preserving residual hearing and supplementing by carefully prescribed hearing aids and lip-reading; *d.* provisions for the social and economic readjustment of the aurally handicapped by local and state agencies.

In his discussion of the "Hard-of-Hearing Problems," Rogers,[70] Chairman of the Special Committee, outlined two independent phases of his report, somewhat after the Burge "Pennsylvania Plan for Tuberculosis." The first was the striking of a balance between case finding, treatment and rehabilitation; and the second, co-ordination of all groups engaged in the fight on deafness.

In 1934 there were five or six Leagues for the Hard-of-Hearing in California — now there are as many as 37 Chapters in the Pacific Zone, more than 30 in California.

The Committee Program for 1938-39 had the following 10 sectional titles:

1. Co-operation of agencies.
2. How to find the hard-of-hearing.
3. Lip-reading and voice-training.
4. The ideal otologist.
5. Audiometers and audiometrists.
6. Diagnostic versus therapeutic clinics.
7. The otologist's place in public health.
8. Hearing aids, single and multiple, and their uses.

9. Fee splitting with hearing aid salesmen and the non-medical audiometrists.

10. Legislation.

The author pointed out that 10,000,000 people in the United States were hard-of-hearing, 3,000,000 were of school age, and deafness was increasing in rural schools as in city schools.

To prevent deafness, he stated that we must first seek for causes and remove them early in the life of the victim. New York had a law requiring annual hearing tests of all school children. Moving pictures were being used in Ohio as an aid to the teaching of hard-of-hearing school children.

Houser, Campbell and Schluederberg,[71] in an attempt to verify the work of Davis and Rommel in the use of prostigmine methylsulfate hypodermically and prostigmine bromide taken orally, in "The Treatment of Deafness and Tinnitus Aurium," studied 56 cases over a period of at least three months. The patients were from 13 to 70 years of age and received 1 cc. of prostigmine methylsulfate (1:2,000) hypodermically every third day; 15 mg. prostigmine bromide were given after meals during the first six weeks of treatment. Audiograms were taken every two to three weeks and local treatment continued as usual. They concluded that in their series of 56 cases of chronic deafness, carefully tested and controlled, prostigmine failed to exert a definitely beneficial effect. They felt that it was doubtful that any real effect was produced on tinnitus aurium. Seven patients felt that some improvement had taken place. None lost tinnitus completely.

Many practical suggestions on "Some Everyday Problems in Otolaryngology" were presented by McMurray.[72] As this practice usually occurred upon a referred clientele, conditions were often complicated by the established impression of the patient as to his symptoms and their cause, as well as the questionable diagnosis from the referring physician.

The indiscriminate diagnosis of sinusitis and the long story of previous treatment was complicating. Another troublesome type was the group of patients who were convinced that they had tuberculosis or cancer.

After discussing in considerable detail the diagnosis and treatment of external otitis, the author attacked the problem of tinnitus, first as occurring from simple conditions. and then from its important relation to progressive deafness.

Evidence has accumulated, according to Selfridge[73] in "A Survey of the Relation Between Nutrition and the Ear," that many conditions of the ear, nose and throat, especially constitutional deafness (nerve and conduction type), sinus infections (allergic or otherwise), and probably enlarged tonsils and adenoids, were related to dietary errors and various growth factors such as endocrine glands, vitamins, mineral salts, amino acids and meteorological factors.

Many diseases supposedly due to infections might prove to be related to nutritional deficiencies, such as beriberi, pellagra, Landry's paralysis, peripheral neuritis including the auditory nerve, gastrointestinal diseases, gall bladder disease, night blindness, fragile blood vessels, rickets. scurvy, pernicious and other anemias, physical deformities, bone and dermal diseases.

The author confirmed his views by the results of the Kerridge examination of 1,000 English school children under 12 years of age, and the United States Health Service Survey in 9,000 individuals in 12 American cities.

In analysis of the nutrition ear, there was obviously an excess of carbohydrates, with vitamins, calcium and phosphorus out of proportion.

Many authorities were quoted by the author, including valuable studies on the VIIIth nerve in relation to vitamins B and C by Covell.

In conclusion, the author commented on the less common vitamins and suggested the value of an optimum diet containing all essential factors which materially reduced the tendency to catching of colds. During an acute infection all the vitamins were essential.

Meller and Arnold[74] reported on a "Clinical Examination of Dr. Zajicek's Hormone Treatment in Cases of Impaired Hearing." The authors invited Dr. Zajicek to personally demonstrate the efficacy of his preparation in the treatment

of 25 of their patients with impaired hearing. Supposedly, the application of the fresh hormone extract on the skin covering the affected organ would result in improved blood circulation and increased supply of oxygen. The ointment was applied to the auricle, the skin covering the mastoid, neck and shoulders. Immediately preceding and after the application, hearing tests were made by whispers and conversation voice, tuning fork and with the audiometer. The youngest patient was age 15 years, and the oldest, age 71 years; there were some cases of otosclerosis included. In no single incident could general improvement be determined with any degree of certainty, and the comparison of audiometric curves with subjective estimates of the patients showed overwhelming discrepancies.

In discussing "Otologic Progress," Lischkoff[75] stated that the general practitioner was keenly interested in deafness from various etiologic sources, but his personal experience in nonsuppurative cases was limited. The treatment of deafness with some, meant inflation and oily nasal sprays; others might be familiar with massage and galvanism, but few realized that a grossly normal drumhead might be seen in a variety of types of deafness.

According to Crowe, there were three common types of hearing impairment: *a.* the type due to a lesion of the conductive apparatus; *b.* that due to a lesion of Corti's organ, or the cochlear nerve; *c.* a combination of conductive and perceptive deafness.

The author stated that in the past we were content to make our diagnosis with the tuning fork but now no one would feel that a test had been complete without the audiometer, the tuning fork and an analysis of the upper respiratory tract.

A new type of deafness has come to the attention of otologists in recent years — aero-otitis, characterized by partial deafness, fullness of the ears and head noises. Passengers on commercial planes were now warned to ventilate the ears by chewing gum, swallowing, yawning, singing, etc.

The use of prostigmine and thyroxine in the treatment of deafness were mentioned, as well as the estrogenic substances recommended by Mortimer and Wright. X-ray therapy and its influence on otosclerosis was mentioned.

The writer mentioned the newer surgical treatment for deafness as proposed by Holmgren, Lempert, Campbell and others, and commented on recent progress on hearing tests as related to hearing aids.

Hayden[76] stated that major hearing losses, and only these, should enter into consideration of "When Hearing Aids Should Be Used," devoid as they are of preventive or curative possibilities. The hearing loss should be enough to handicap the individual educationally, socially and economically. The otologist's study, with a review of the patient's history, physical examination, careful audiometric check-up and tuning fork tests, should precede the prescribing of a hearing aid.

There were no hard and fast rules to be laid down, but an average hearing loss of 25 to 40 dcb. would warrant the use of a hearing aid. This depended on the character of the loss and the age of the patient. Air and bone conduction receivers were prescribed to suit the individual case, whether the greater loss was for air or bone conduction.

Tube aids gave straight line amplification and consequently adapted themselves to regular losses; carbon aids more readily adjusted distorted losses, as their amplification must be confined to the auditory area — 300 to 3,000 cycles.

The audiogram indicated which ear was to be fitted. When the air conduction loss was greater in one ear than the other, generally the ear with the greater loss should be fitted, if a substantial improvement could be obtained.

When the bone conduction loss was the same in each ear, fitting the ear that had the greater overall loss (bone and air) usually gave the best result.

Under the title of "Ears," Foster[77] pointed out that children often failed to realize that they had hearing handicaps and that when they have never been able to hear normally, they had no general basis for comparison with other children. Some children developed a dull facial expression and progressed slowly in school when their hearing was impaired. She discussed the general anatomy of the ear and stressed the way children should blow the nose, in order to prevent trouble from ascending the Eustachian tube to the middle ear. She

stated that the following symptoms of ear difficulty should be regarded as suspicious:

1. Deafness, dullness, heaviness or a blocking sensation in the ears, persisting for more than a few minutes, particularly after exposure to cold, swimming, head colds, fatigue, etc.

2. Hearing better than others in noisy places.

3. Marked language retardation. Failure to acquire a considerable vocabulary by three years of age.

4. Asking frequently for a repetition of words and phrases.

5. Confusion of similar sounds; one of the first signs of impaired hearing.

6. Retardation in school.

7. Constant buzzing in the ears.

8. Dizziness.

9. Failure to respond when called or to properly locate the source of sound.

10. Moisture, discharge or odor from the ear canal.

Hearing tests and proper care during an acute infection would do much to eliminate deafness in children. "Procrastination was the thief of hearing."

GROUP 3. MECHANICAL AND SURGICAL PROCEDURES.

In "The Present Trend in the Treatment of Progressive Deafness," Canfield[25] reviewed surgical fistulization of the otic capsule from Passow in 1897 to the present time, and stated that great strides had been made in the past 20 years in correlating auditory acuity with the actual lesions of the cochlea and nerve paths. He further stated that we could no longer be dogmatic about designating a nerve deafness if the high tones were affected, and an obstructive deafness if the low tones were diminished.

Along with the intense interest in the fundamentals of hearing, the clinical phase had been developed and accumulated knowledge applied to patients. Great strides had been

made in radio, sound transmission and electric hearing aids. Some otologists felt that they were the best means for improving hearing, but such apparatus still lacked the ability of the normal ear to maintain automatically the normal proportion of sound with an increasing distance.

A few otologists were attacking the problem another way. Histologic work had shown that in many ears the cochlear mechanism remained intact, and only because sound energy did not reach the cochlea to be transferred into electric energy had the ear partially lost its microphonic ability.

Holmgren, Sourdille and Lempert had devised and used techniques with varying success, in attempting to produce permanent fistulae in the semicircular canals. Selection of case for this operation was not difficult. Integrity of the cochlea and neural mechanism was essential. When bone conduction was normal for the critical frequencies of 256 to 2,048 cycles per second, the neural mechanism could transmit the human voice. If bone conduction was decreased, the neural mechanism might be intact, but at present this could be determined clinically. If we selected those whose hearing by air conduction was decreased but whose bone conduction was normal, we could include them in the group whose air conduction would be improved by labyrinthine fistulization. Age, occupation, general physical condition and, most important, the psychological reaction of the patient must be considered.

The term, otosclerosis, should be used only to designate a pathologic entity and not as a clinical diagnosis. It was a disease of bone occurring in and around the otic capsule and, incidentally, involved the hearing mechanism.

This might fix the ossicular chain and interfere with airborne sound reaching the cochlea. Sometimes this process invaded the bone of the modiolus and the osseous spiral lamina, thereby affecting the cochlear nerve. When this occurred, bone conduction was decreased and the operation was of little or no value.

The author reviewed his experiences while working on the subject with Holmgren. He stated that Holmgren had operated on more than 100 patients without a fatality, using a method which consisted of producing a fistula in the external

semicircular canal at its accessible portion in the floor of the aditus, by a postauricular transmastoid approach. The great problem had been to find some type of tissue which would not permit or encourage bone proliferation to fill in the fistula. The best method for this had not been determined. A number of foreign bodies had been implanted without success. Electrolysis of bone in the presence of certain metals had been found to be a cause for nonunion of fractures of the long bones in which metal plates had been used for fixation. The efficacy of this had not been tested so far in this work. A radium preparation was tried in the newly prepared fistula of the monkey. After seven days it was removed, and the animal allowed to live for four weeks. Temporal bone sections were studied by Prof. Nager, and in the region of the fistula not only was there *no* bone regeneration but the surrounding bone appeared to be undergoing devitalization. The biology of bone was still a matter requiring far better knowledge of its intricate processes.

Hughson's procedure of round window graft was described and the author felt that this was progressive work, and stated that some of Hughson's patients believed they heard far better than was indicated by the audiogram.

He also described Wittmaack's operation for changing the blood supply to the otic capsule, which he did by elevating the dura around the petrous apex in the hope of obliterating hypothetical venous stasis. Canfield, after visiting Wittmaack's clinic, was unconvinced that this procedure had merit. The claim for this operation was not an improvement in hearing but a cessation of the pathologic process.

He stated that thyroxin, metabolic management and endocrine therapy had not produced anything of significance in the treatment of deafness.

He concluded with the statement that the picture of chronic deafness was not quite as dark as it was 10 years ago — hope could be offered which a few years ago was impossible.

In his article, "Critique of Surgical Treatments for Deafness," Fowler[79] stated that recent reports on the surgical treatment of deafness were open to criticism because of their optimistic conclusions and the poverty of facts supporting

these conclusions. The whole problem of how to treat deafness was immeasurably complicated because we knew so little of the causes. Deafness was only one symptom of trouble in a very complicated sense organ. This author questioned the accuracy of present methods of the testing of hearing and said that the more we tested a patient the better his hearing might appear, no matter what the treatment.

Some surgeons claimed that they did not care what mechanical tests showed — they claimed that when the patients said they heard better, the treatment must be worth while. These surgeons forgot that patients were suggestible and that any procedure would make them think they heard better. In the Vanderbilt Clinic there were a number of patients who said they maintained a better hearing on biweekly injections of 1 cc. of normal salt solution. At this clinic and the Manhattan Eye and Ear, there were dozens of patients who thought they heard better with prostigmin, insulin, tuberculin, vitamins and operations, but properly taken audiograms did not substantiate their statements. The success of almost all the heralded "cures" for deafness was based upon improvement of the patient's general physical and mental condition. If the patient felt better, he listened better and heard more. No article on the treatment of deafness was worth the paper it was printed upon, if it did not contain detailed multiple audiometric records taken before and after particular therapy was begun. Certain frequencies were very much more important than others. On a basis of 100, 256 could be weighted as 3; 512 at 7; 1,024 as 40; 2,048 as 40; and 4,096 as 10.

In regard to the surgical techniques for the improvement of hearing, the author stated that Wittmaack and others had demonstrated that once the nerve or end-organ of hearing was destroyed, there was no regeneration; accordingly, the only types of deafness amenable to surgery were the conduction types which developed from otosclerosis or otitis. He classified the procedures broadly under the headings: preventive, palliative and specialized techniques. Under *preventive,* he listed such procedures as myringotomy, or simple mastoidectomy, and under *palliative,* he listed simple, modified radical and radical mastoid operation in cases of long-standing infection, because experience had shown that if suppuration continued, deafness on the average increased

from year to year, especially in children. The radical and modified radical operations improved hearing in 30 per cent of the cases, made no change in 37 per cent, and the deafness was made worse in 33 per cent. Under *specialized techniques*, he described Hughson's operation of plugging the round window with a fascial graft. Analysis of his careful and thorough papers showed there was often much improvement in the other ear, and that the improvement from operation tended to fall off as the months went by, but he reported his cases in detail and invited criticism, and his papers should stand as models for all those working in the field of deafness.

The author mentioned Dixon's operation of placing a roll of skin in the mastoid so as to produce a secondary Eustachian tube. Multiple accurate anteoperative and postoperative audiometric records were necessary before accepting any procedure which is theoretically so unsound.

The author discussed in great detail fenestration of the labyrinth, or the popularly known "window operation." He stated that so far the most promising surgical attack on conduction deafness was found in the methods of Maurice Sourdille. His technique consisted, in essence, of turning the outer surface of the medial end of the superior and posterior membranous canal wall back over a fistula in the horizontal canal. In order to get the thinnest part of the flap and to shut off the middle ear from the mastoid cavity, as well as to produce an altered tympanic system, using the incus as a scaffold, he cut off the head of the malleus, permitting the whole upper part of the drum membrane, with its attached meatal flap, to be moved mesially. The author saw 39 cases operated on by this technique in 1937, and 14 in 1938 — one case of eight years' standing, two of six years, one of five years, and several of two, three and four years' standing. In all but seven of the 53 cases the fistula was active.

The Lempert technique used in this country was a modification of the Sourdille. The incision was made endaurally, and he added the use of the dental drill and polishing burr to the armamentarium of the operation, and he had made it a one-stage operation.

Fowler raised the question, "Why do labyrinthine fistulae close?" In answer, he said that bone chips tended to act as a

foci from which new bone growth took place. He suggested the use of high magnification, such as a Zeiss distance lupe, with 6 X magnification, to keep the wound free from bone dust. Nager has pointed out that in fractures through the labyrinth, it was only from the periosteal capsule that bone regenerated. He suggested that if the periosteal bone could be prevented from growing back over the fistula it would always remain open; since the endochondral bone was very thin over the periphery of the horizontal canal, the fenestra should always be made in the solid angle where the periosteal capsule was relatively thin. The size of the fistula appeared to be important. The larger the fistula the less likely it was to close.

Infection tended to speed bone regeneration, so a scrupulously sterile technique was imperative in this work, not only in operating but in dressing these patients.

The dangers of the operation, he pointed out, were infection of the lateral sinus wall, labyrinthitis and meningitis. No cases of meningitis had been reported as yet.

As to what to expect from the operation: — no one knew exactly what percentage of cases could be expected to have satisfactory improvement, nor how long it would last.

Alternatives to the operation were lip-reading and hearing aids.

In conclusion, he stated that up to the present the reports which he had examined, which were complete enough to analyze, showed that less than one-third of the cases operated upon had obtained and maintained satisfactory improvement for more than six months. He felt that too much experimental fenestration had been done on humans.

Hughson[80] presented "An Appraisal of Four Years' Experience with Round Window Grafts for Deafness." Previous reports of these cases had been made, but in confirmation of the reported benefit the present paper presented an analysis of the still relatively small series of 36 operations.

These cases were studied over approximately a four-year period, the oldest for three years and nine months, the youngest for six months. Preoperative tinnitus was reduced in five

out of 15 cases but never cured. One case of postoperative tinnitus developed. In 60 per cent of the cases reported there was subjective improvement in hearing.

The author referred to the fact that no audiometric improvement of the threshold of hearing, representing less than a sustained level of 10 dcb., could be regarded as of real consequence.

The range of improvement must be extended both below and above the critical frequency range of 512 to 2,048 cycles.

During the operative period, while the drum membrane remained open, 20 per cent of the patients had substantial gain; 80 per cent, impairment. Of the patients operated, 21 were female and eight male. Decade enumeration was as follows: second decade, three; third decade, five; fourth, 11; fifth, 10; sixth, none; and seventh, one.

The original determination of 50 as the maximum age was confirmed by results.

A statistical analysis of four years' experience demonstrated a trend toward improvement in hearing, with no ultimate damage likely to occur from the operation.

In "Operative Treatment of Otosclerosis. Reply to a Recent Criticism," Shambaugh[51] has made a frank reply to the Guggenheim position on "artificial fistula in cases of otosclerosis." This discussion concerned the following paragraph:

"The treatment of otosclerosis by means of: *1.* reversal of halisteresis, and *2.* regeneration of the damaged neural mechanism through dietary regulation and the administration of calcium, phosphorus and vitamins, has, after three years of experimentation, succeeded in producing improvement in hearing far greater than has any fistula operation thus far reported."

According to the author, there have been no substantial improvements in hearing in cases of otosclerosis from any treatment, medical or surgical, as great as those reported after the Lempert operation. All critical observers agreed that deafened persons were easily stimulated by hope induced by any form of treatment, commenced to pay greater atten-

tion and, therefore, seemed to hear better. This apparent improvement was psychologic rather than physiologic.

The author further claimed that no treatment for deafness could be accepted unless repeated audiograms, before and after treatment, showed a maintained improvement of at least 10 dcb., including the conversational frequencies, 512, 1,024 and 2,048.

Thirty-one cases with carefully repeated audiograms were reported, which were open for examination and hearing tests.

Goodyear[52] described a "Postauricular Approach in Operative Treatment of Otosclerosis," in which the performance was simple and an excellent opening was obtained in the external horizontal canal. This secured the thinnest and most approximate membrane which could reasonably be obtained to cover the opening made in the canal.

Under local or general anesthesia, a postauricular incision was used, and carried well posteriorly to the edge of the hair line, and not below the tip of the mastoid. The cortex was removed and the horizontal semicircular canal and short limb of the incus were brought into view by means of the Spratt curette. The soft tissues were carefully separated from the outer portion of the posterior bony wall of the canal. When the posterior soft tissues were exposed in the outer two-thirds, a narrow scalpel was used to incise the soft tissues of the posterior wall of the canal through and through at right angles to the canal, 1 cm. from the posterior attachment of the tympanum. The outer part of the canal could now be moved without danger of any detachment of the annulus tympanicus. The rest of the bony posterior wall of the canal was removed from the soft tissues by means of the Richards mastoid curettes. When the horizontal semicircular canal was exposed, it was uncapped by a small, sharp Richards curette. The canal flap was then placed over the opening.

The author reported two of his own cases, in both of which the middle ear was completely sealed off.

Canfield[53] has endeavored to present the clinical picture of patients with "The Type of Deafness Which Responds to Fistulization of the Otic Capsule," and has discussed the neces-

sary findings for patients to be included in this group, and the extent of deviation still offering promise.

The present theory was that a new opening into the otic capsule permitted sound waves to produce an effect on the cochlear fluid, stimulating the organ of Corti apparently by mechanical force. The author believed that a vibrating basilar membrane was necessary to stimulate the hair cells, and that the mechanical process rearranging the ear with a new fistula neither improved the microphonic effects of the cochlea nor the neural mechanism transmitting the impulse to the brain. With the annular ligament fixed, less movement of the endolymph resulted, with less movement of the basilar membrane and the round window. With the third opening into the bony otic capsule, whose oval window was fixed, there was new possibility for endolymph movement, and it was supposed that the fistula provided that vibratory movements of the endolymph were greater for the same volume of sound pressure.

Briefly those could be helped whose lesion prevented sound impulses from reaching the organ of Corti but whose nerve and cochlea were intact. The point at issue was how could these patients be selected.

The author then discussed the patient's deafness in relation to conversation, relation to conductive type of deafness and evidence of perceptive lesion. The progression of deafness was then discussed and the fact that fistulas were only of service in absence of ear infection.

After discussion of influential factors in the patient's general background, including hereditary deafness and drug influence, adjustment to social situation and desirability of hearing aid, the author presented one of his own cases of fistulization followed by improvement, and two cases without improvement. The author maintained that in final interpretation before operation was advised, at least three audiograms must be made for an accurate preoperative level.

In his summary, the author stated that the overwhelming evidence was that after a fistulae is made to the otic capsule of a partially deafened person, the internal ear has been rearranged so that a sound of certain intensity could stimu-

late the organ of hearing, which before the fistula was inaudible. The author considered, further, that the cases which had the best chance of improvement were those in which the sound waves were obstructed in their passage to the organ of Corti, while this structure and the remainder of the neural mechanism stayed normal and intact. Although some reports indicated that ears with a defect in the neural mechanism had also been improved by fistulization, further experimental study must determine their place in the predictable group.

According to Maxwell and Richter,[84] in "Radical Mastoidectomy; Its Effect on Hearing," the surgeon must discuss with his patient before operation the following: *1.* the dangers involved in chronic suppuration; *2.* the danger of impairment of the facial nerve; *3.* the probability of postoperative freedom from all discharge; and *4.* the possible changes in hearing.

After a brief review of the literature, the authors concerned themselves chiefly with the question of hearing after a radical mastoidectomy. The following conclusions were drawn:

1. Preoperative loss of hearing had been determined in large degree relatively clearly in the chronic suppurative process.

2. A poor prognosis for residual hearing should be given to persons over 45 or 50 years, to those having infection of the middle ear and mastoid of short duration, and perhaps to those who had comparatively good preoperative hearing, as indicated by an average loss in the critical frequencies of 25 to 30 dcb.

In the consideration of radical mastoidectomy with clear indications, the possibility of decreased residual hearing should not be a deterrent. The chances of slight improvement or slight impairment of hearing were about equal, with an average of less than 10 dcb.

Herrmann[85] discussed the "Diagnostic Value of the Oto-Audion in Case of Deafness, with Normal Tympanic Membrane." He stated that tone tests were made either with tuning forks or by electrical means. Qualitative and quantitative tests were formerly done with Bezold tuning forks. The audiometer had been found to be a better instrument for

quantitative tests. It also made possible bone conduction tests in all frequencies. Testing took less time with the audiometer and the progress of therapy could be checked with more accuracy. The oto-audion should be used in the following conditions:

1. Where difficult diagnostic results were to be computed with great precision.

2. Where expert opinions were required on the question of heredity.

3. Where decisions must be made as to therapeutic measures and auditory possibilities.

In "Lucae's Sound Instrument in Cases of Auditory Murmurs and Deafness," Goebel[86] deplored the fact that this instrument which was introduced about 60 years ago, was apparently almost forgotten today, being merely a name to most otologists. He stated that for treatment with the sound probe the following characteristics were essential: *a.* moderately strong tonicity of muscle; *b.* delicate sensitivity to slightest pressure with minimum movement; *c.* uniformly strong suggestive muscle contractions following each other almost imperceptibly.

According to the author, treatment was facilitated if the end of the probe was wound with a pledget of cotton; the pressure was more relaxed and distributed over a somewhat greater surface. The point of insertion was the end of the handle of the malleus, and the number of pressure thrusts at one treatment varied. The upward course of the probe must be very gradual, and it was important that the movement must be stopped at the moment when even the slightest resistance was noted. Careful observation should then be made of the temporal region in the orbits, and if distortion was perceptible, pressure should be reduced.

Although treatment was short, two minutes, it was taxing, especially for the beginner, since it was necessary to be tensely alert. In new cases, after the initial treatment, remarkable improvement in hearing was often noted, due to mobilization of the stapes. Such typical experience led one to conclude that properly proportioned treatments of auditory disturbances were inconceivable without Lucae's pressure probe.

Each practical thrust at the handle of the malleus shifted the stapes inward. As the posterior part of the annular ligament resisted further shifting, the anterior part of the stapes turned inward.

Lempert[57] explained his technique in "Endaural Fenestration of External Semicircular Canal for Restoration in Cases of Otosclerosis; Summary Report of 120 Cases," as the following:

1. Creation of troughlike fenestra in the bony capsule of the external semicircular canal by a polishing and burnishing burr;

2. Incorporation of this within the confines of a newly constructed air-filled and pneumatically sealed tympanic cavity;

3. Reconstruction of the osseous portion of the external auditory canal, permitting air-borne sound to reach the new inner wall of the external auditory canal.

Enumeration of the refinements of technical minutiae included the control of bleeding, complete cell exenteration of the mastoid process, opening of the incudomalleolar joint and removal of the head and neck of the malleus, removal of the Eminentia Pyramidalis, decompression of the dura in the epitympanic space, evacuation of the troughlike fenestra, treatment of bone dust and placement of the temporomeatal membrane.

The author enumerated seven indications for fenestration:

1. Bilateral and progressive loss of hearing.

2. Fixation of stapes in the oval window; normal mobility of the round window membrane.

3. Loss of hearing by air conduction in the conversational frequencies, 512, 1,024 and 2,048.

4. A normal and intact tympanic membrane.

5. Absence of middle ear infection.

6. Patent Eustachian tubes.

7. Normal state of health.

These surgical accidents were discussed:

1. Injury to the cutaneous tympanomeatal membrane.

2. Perforation of the membrana tensa.

3. Fracture of the vertical mastoid portion of the Fallopian canal.

4. Disarticulation of the incudostapedial joint.

5. Fracture of the sinus plate.

After discussing the various possible postoperative sequelae, the author summarized 120 cases during the last two years: Practical physiological hearing was restored in 69 cases; some improvement was noted in 10 cases; further impairment in 14 cases, and hearing unimproved in 27 cases.

This presentation has been accompanied by many illustrations and diagrams of audiometric readings; comments on osteogenesis and clinical observations on fenestra vs. hearing aids have been freely discussed. The writer has felt that there was no surgical risk to life under strict rules of asepsis, and that hearing had been permanently improved in 80 per cent of properly selected cases.

In his "Summary of Round Window Graft Operations Performed for Deafness," Hughson[88] stated his belief that one function of the round window and membrane was to dampen the intensity of acoustically induced stimuli reaching the perceptive mechanism of the inner ear.

At Johns Hopkins, experimental fixation of the round window membrane by pledget of cotton firmly pressed in the niche, produced improvement of 10 to 30 dcb. in hearing acuity of normal ears as measured by changes in electric potential, either in cochlea or auditory nerve. Whispered tones were particularly clearly heard and high frequencies had increased intensity.

In animal experimentation, various kinds of living tissue, fascia, muscle and periosteum, were used in place of cotton.

Experimentation led to the belief in the safety valve protection of the round window membrane. The following procedure was advanced: Postauricular and mastoid approaches

were discarded and a simplified technique of direct approach through the canal and tympanic membrane was developed. The canal wall was sterilized by instillation of 70 per cent alcohol, then ether. Under a magnification of from three to four diameters, and adequate illumination, an incision was made along the posterior border close to the annulus, a flap was turned down and the promontory and round window niche were visualized. The graft was packed firmly in position and blood was carefully removed from the middle ear. The closure of the tympanic membrane usually occurred in two weeks. Fifty years should be the upper age limit, sex was unimportant, but surgery would be futile in a 50 dcb. hearing loss.

The operation was performed under avertin anesthesia with a hospitalization of three days, and the poorer ear was always selected for the first operation. An intact tympanic membrane was a prerequisite for the round window graft operation. Fixation of the stapes footplate and mobility of the tympanic membrane were not greatly important. No tympanic membrane failed in subsequent healing. While emphasis was laid upon the three critical frequencies, 512, 1,024 and 2,048, the octaves immediately above and below showed favorable reaction to therapeusis. Tinnitus was never completely eliminated.

Summary: In 35 control cases, no infection nor rupture of the round window membrane was noted and the graft always remained fixed. Fulfillment of hearing improvement standards had been obtained.

In the opinion of Campbell,[89] suitable cases of conductive deafness could be helped by fistulization of the labyrinth, with a lessening of tinnitus, but permanent value would depend upon an open fistula. He reported on "Results in the Labyrinth Fistulization Operation for Chronic Progressive Deafness."

The author stated that a favorable result would depend on three factors: *1.* proper selection of patient; *2.* proper operative technique; and *3.* proper postoperative care.

In the proper selection of the patient, the degree of deafness, status of the bone conduction, adequacy of the vestibular

function and state of the canal wall, drumhead and middle ear were vitally important.

According to the author, perforation did not necessarily interdict operation, and it was possibly true that the best results might be obtained in typical cases of otosclerosis with stapedial fixation.

Under operative technique, the author counseled the preservation by scrupulous care of the cutaneous membrane lining the posterior and superior walls of the external auditory canal. The avoidance of dislocation of the incus, requiring its removal, or laceration of the tympanic membrane, was a delicate point of technique. The making of the fistula and fitting of the temporomeatal cutaneous membrane over it also required careful technique.

In postoperative care, three important suggestions were made: *1.* asepsis must be maintained; *2.* excessive granulations must be treated; and *3.* general care of the patient must be taken.

The author made a detailed report of eight cases which had been operated by the Lempert technique. In analysis of these cases, three had gained improvement in hearing, the first retaining 21.4 per cent improvement after 14 months; the second, 20.3 per cent improvement after a year; and the third, 21.9 per cent improvement after eight months.

The author reported two cases of prompt closure with bony regeneration; after reopening, one had a secondary closure of the fistula, but the other maintained a fairly active fistula with 5 per cent improvement in hearing nine months after operation.

In two of the cases, hearing was made worse. Both were evidently unsuitable for operation, one with decreased bone conduction, and one with a dead labyrinth.

The remaining case had a previous radical mastoid operation and the hearing had shown slight loss over the preoperative status.

Under the title, "Surgical Treatment of Deafness," Lischkoff[90] presented a historical review, commencing with the simple myringotomy with hearing improvement, cited the

removal of the stapes by Kessel in 1876, trephining of the promontory by Passow with temporary improvement in 1897, covered the early work by Bárány and Jenkins, and discussed more carefully the fistulization methods of Holmgren. The author concluded with a rather intimate review of the contemporary workers in the fenestration field. He stated that the final estimate of fenestration had not been made.

According to L. and P. Guggenheim,[91] otosclerosis was not only an osseous dystrophy producing an obstruction to sound conduction, but also a disease of the VIIIth nerve producing perception deafness, and an "Artificial Fistula in Cases of Otosclerosis," even though successful, could not inhibit further deterioration of the neural mechanism.

The authors stated that "the treatment of otosclerosis by: *1.* reversal of halisteresis; *2.* regeneration of damaged neural mechanism by dietary regulation and administration of calcium, phosphorus and vitamins, has after three years' experimentation succeeded in producing improvement in hearing far greater than has any fistula operation thus far reported." They believed that analysis of diet and a knowledge of biochemistry and vitamins would be the ultimate solution.

A case was reported in which an artificial fistula, patent and with positive reaction of vertigo, still proved incapable of transmitting sound. The authors came to the conclusion that the complexities and difficulties of the present fistula operation had no point whatever.

They reported research work on the results of making fistulae with sharp chisel and with burnishing burr and electrocoagulation. Two divisions of this work were reported: *1.* the status of the fistula 18 days after operation; and *2.* conditions two months and two days after operation. These two procedures, amply illustrated, were published in detail.

In summary it was found that after 18 days a lively and widespread new bone formation existed when the fistula had been made with a sharp instrument or burnishing burr. No evidence of osteogenesis was seen at this time when the burr had been followed by osteodesiccation by means of electrocoagulation.

After two months and two days all osteoblastic activity had ceased and all mesenchyme had been replaced by fatty

marrow with islands of blood forming cells, or by fibrous marrow. The most patent fistula resulted from use of the sharp instrument; next, that with the burnishing burr and electrocoagulation; with the burnishing burr alone, the largest amount of new bone formation occurred.

In conclusion, the authors failed to substantiate Canfield's conclusion that the use of the burnishing burr was not followed with new bone formation. The fistulas here reported were, as in Canfield's experiments, not in the labyrinthine capsule but in the membrane bone of the skull. Much further research was necessary in the neuropathy of otosclerosis. The authors considered the time-honored posterior approach far superior.

According to Nadoleczny-Millioud[92] in "Artificial Tympanic Membrane in Comparison with Hearing Apparatus," the artificial tympanic membrane was used only in defects of the tympanum, or after radical operation, when the hardness of hearing was bilateral. It was hardly ever employed while one ear still had adequate hearing. It had the disadvantage of making the patient dependent on the otologist to change or readjust the membrane frequently. Moreover, he had to make and fit it, because the tympanic prostheses obtainable on the market were rarely suitable. He stated that occasionally the artificial tympanic membrane irritated and produced secretion but that this was harmless and disappeared quickly, and the fear of recurrent suppuration was generally exaggerated. It should not prevent a trial with the tympanic prostheses in cases in which they were indicated; *e.g.*, in all dry perforations or scars.

He reported one disadvantage in common with the hearing apparatus; noises which the patient did not hear before were now excessively loud. Because of this, the patient at first might find it difficult to go to sleep.

Its disadvantages were: *1.* not visible from the outside; *2.* caused practically no distortion; *3.* did not make the patient more hard-of-hearing for some sounds. The available electrical hearing devices did not transmit undistorted sounds. Another advantage of the artificial tympanic membrane was the disappearance or weakening of subjective sounds (tinnitus).

He stated that there were two methods of estimating the value of a hearing prosthesis: *1.* the exact quantitative and qualitative examination of the auditory capacity, with and without the prosthesis; *2.* the reports of patients who had some knowledge of acoustics and of music, and who observed themselves carefully.

The author described in detail the experiences of one patient who had undergone a bilateral radical operation and who had used not only several electrical hearing devices but also an artificial tympanic membrane of silver-leaf. The patient asserted that the acoustic effect of the silver-leaf membrane was comparable, although not quite equal, to that of a rather large and cumbersome (weight, 9 kg.) condenser-microphone, in which the loudness could be modified and which he used when attending meetings and lectures. The subjective effect of the artificial tympanic membrane, however, was even better than that of an apparatus. It overcame all self-consciousness because it made strings and technical devices unnecessary. The patient had no difficulty in keeping up a conversation in a small group when the membrane fitted well. The ability to hear everyday noises had a beneficial psychologic effect. The superiority of the artificial tympanic membrane over other hearing aids was due chiefly to the faithful transmission of the high tones up to the fifth-marked octave. Artificial membranes were now being made not only of silver-foil but also cellophane. The fact that this hearing prosthesis was suitable for only certain patients was, in the belief of the author, no reason for neglecting it completely.

Meyer[93] discussed "A Possible Explanation of the Efficacy of Suboccipital Puncture in Degenerative Deafness, with or without Otosclerosis." According to the author, the procedure of withdrawal, at one operation, of 35 to 50 cc. of cerebrospinal fluid by suboccipital puncture brought about an improvement in hearing in a certain proportion of cases of otosclerosis, as well as in those of degenerative disease of the labyrinth. Since it was impossible for the physical factor of change in pressure to produce the observed results, it could only be some biological influence which had caused the change. According to the present day theory concerning the rôle of the fluid in the inner ear, it was very probable that in certain grades of the hypotoxic condition of the

ductus cochlearis, and especially of the organ of Corti, it was possible to approach the normal anatomic physiologic condition of these tissues by reduction of the pressure of the perilymph.

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PLASTIC BY TRACHEOSTOMY.

DR. E. I. MATIS, Kaunas, Lithuania.

Inoperable growths, chronic cicatricial stenosis of the larynx, etc., often require the wearing of a permanent tracheal tube. For this purpose, the usual procedure of tracheotomy is inconvenient because the wound heals slowly. In such cases tracheostomy is preferable, but the typical tracheostomy described by Sebileau, Collet and others has the disadvantage that not all parts of the wound are covered with skin. Especially do the upper and lower edges of the tracheotomy wound remain open, and it is from there that the spread of granulation comes. With the aid of a very simple plastic it is possible to establish a condition by which the whole wound is covered with skin, thus creating a situation for good healing.

The operative technique is as follows:

1. Position of patient, as usual.

2. Local anesthesia, as usual.

3. Two incisions in the form of an "X" are made in that part of the throat corresponding to the opening of the trachea (see Fig. 1.)

4. The preparation of four triangular flaps of skin undermined to a suitable distance from the incision. These flaps of skin can be advanced on all sides to cover a defect of considerable depth (see Fig. 2).

5. The liberation of the anterior wall of the trachea.

6. An oval tracheostomy opening is made (see Fig. 3).

7. The edges of the triangular flaps of skin are sewn to the tracheal incision in order to allow the epithelium to grow inwards and to line those parts of the tracheostomy wound that are devoid of it (see Fig. 4).

The tracheotomy tube is inserted.

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This method can be used for both superior and inferior tracheotomy. With superior tracheotomy the incision and the flaps may be smaller as the trachea lies nearer the surface. In inferior tracheotomy, however, it is necessary to

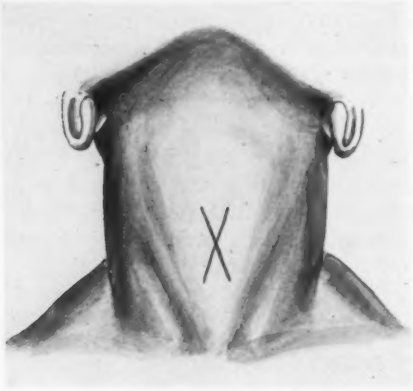

Fig. 1. X-shaped incision.

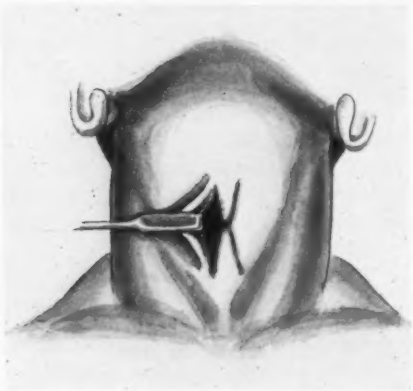

Fig. 2. Forming four skin flaps to cover the tracheostomy wound.

make the flaps larger and more mobile. Special attention must be given to the lower triangular flaps of skin as in this location the trachea lies deeper.

Certain technical difficulties arise where the trachea lies

very deep and the neck is short and thick. In such cases it is necessary to increase considerably the length of the incisions and to free the skin widely from the underlying tissue. In order to decrease the tension of the trachea-skin suture,

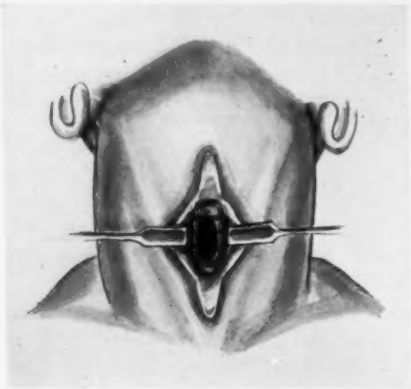

Fig. 3. Oval tracheotomy opening is being made.

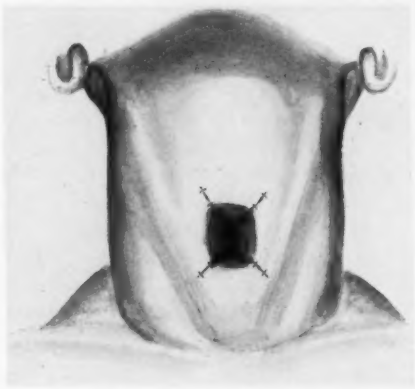

Fig. 4. Operation completed.

the use of a special stitch so as to distribute the tension is advised.

With the aid of the above-mentioned plastic of the triangular flaps by tracheostomy, the following results are obtained:

The whole wound, including the edges of the tracheal opening, is covered with skin; thanks to which, healing is quicker. There is also less chance of further infection and less danger of future stenosis or complications on reintroduction.

This method is also indicated in those cases where a permanent tracheostomy opening is desired, as in inoperable growths of the larynx or chronic inoperable stenosis of the larynx. This method can be used where especially favorable conditions of the wound are desired; *i.e.*, tuberculosis of the larynx with stenosis.

THE EXPERIMENTAL ADMINISTRATION OF SULFAPYRIDINE TO GUINEA PIGS INFECTED WITH HUMAN TUBERCLE BACILLI.*†

DR. FRANK R. SPENCER, DR. RICHARD W. WHITEHEAD and GERALD J. DUFFNER, B.A., Denver.

The cultures used in these experiments were made from a human sputum isolated strain. The strain was of such a virulence that 0.000,010 mg. of the moist growth would cause the death of a guinea pig in three months. The bacilli used in this work were grown on Petroff's media, and in each experiment the culture was three weeks old when used. The inoculating procedure used is the one developed and used by Corper[1] at the National Jewish Hospital for Consumptives in Denver. It is as follows: 10 mg. of the moist growth from the culture tube is carefully weighed in a centrifuge tube. This is then ground against the side of the tube, using a glass rod. While doing this, several drops of a 0.5 per cent sodium taurcholate solution are added to facilitate the grinding of the colonies. After this is complete, the suspension is made with 0.9 per cent NaCl solution. Successive dilutions are then made until the desired strength is obtained.

The sulfapyridine used in these experiments is the pure crystalline compound obtained from Merck and Co. The drug was administered to the animals, suspended in a 20 per cent weight. This dosage was decided upon because Birkhaug[2] has acacia solution. The dosage used was 0.2 gm. per kg. of body worked out the concentration curves in the various body tissues for this dose and found them within the range used for clinical therapy.

Guinea pigs were used for inoculation and an equal number were used as controls. The tuberculous lesions were allowed to develop before sulfapyridine was administered. The drug was withheld until the lymph nodes enlarged. The

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drug was first administered by a stomach tube. Later it was given in the drinking water.

Many of the animals died. These showed at autopsy an extensive tuberculosis, especially in the untreated animals.

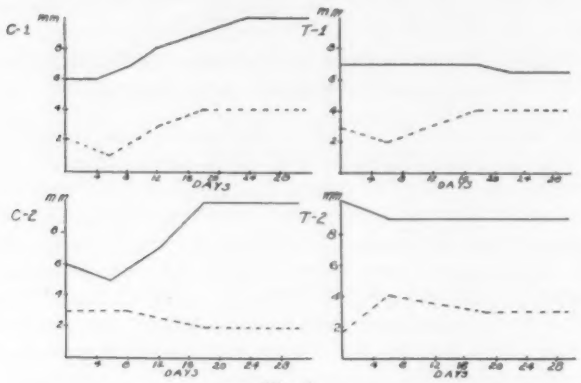

Fig. 1.

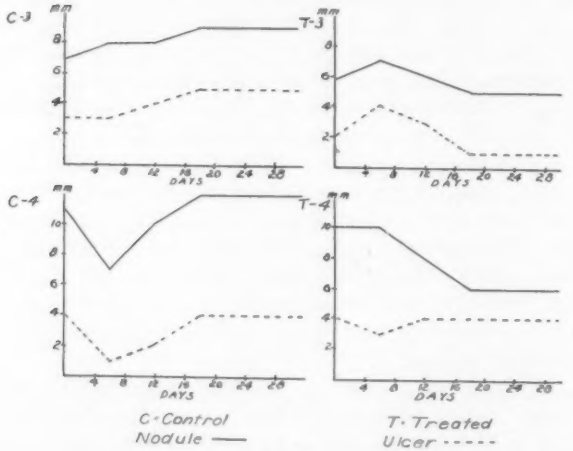

Fig. 2.

In the treated animals which survived there was some slight improvement, but not enough to justify very much optimism.

We encountered the same difficulty which most investigators have when we tried to get a drug to penetrate a bacillus

the content of which is 40 per cent fat. We will continue the experiments with sulfapyridine and other drugs. Sulfanilamide has been tried by other investigators and found to be of no value. We, therefore, have not used this drug. We have in mind a method of administration which may enable the drugs used to penetrate the fat-laden tubercle bacillus. We hope to report better success with this later on. Chemotherapy has yielded such brilliant results with the streptococcus and the pneumococcus that it seems only reasonable to expect to find a chemical which will destroy the tubercle bacillus.

Feldman and Hinshaw[3] administered sulfapyridine to guinea pigs before the pigs were inoculated with tubercle bacilli and continued the administration of the drug after inoculation. They found that sulfapyridine exerted a definite and striking modification and retardation of the expected course of the experimental tuberculosis in guinea pigs. Our experimental work so far has been confined to treatment of the guinea pigs after the tuberculous lesions have been given time to develop before any sulfapyridine was administered.

The accompanying chart shows the progress made by the controls and by the treated animals. This graph shows the average results for four controls on the left side and for four treated animals on the right side. We have used to date 60 guinea pigs. The treated animals are better than the controls, but the tuberculous lesions have not been cured as we had hoped they would be. The continuous line shows the progress of the nodule and the broken line shows the progress of the ulcer.

We are indebted to Mr. Glen E. Mills, the photographer at the medical school, for his valuable help with illustrations.

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THE INDICATIONS FOR SURGERY IN MENINGITIS.

DR. EDWARD KING, Cincinnati.

The otorhinologist has well defined principles to follow in uncomplicated cases of mastoid and sinus infection. He is naturally loath to deviate from the recognized indications in these cases because experience has revealed that disastrous results follow what he is pleased to call meddlesome surgery. He knows that the mastoid operation is safer and followed by good results if the infection is well localized, and he shrinks from attacking an acute sinus before walling off has taken place.

At times a threatened or an already established invasion of the meninges occurs and the otorhinologist is confronted with a serious problem. I suspect that there is very little difference of opinion when such a situation presents itself as to the course of action. Experience has pretty definitely shown that early and bold surgical measures must be instituted if surgical therapy is to be of any value.

The incidence of meningitis complicating middle ear infection is by far more common than sinusitis. Courville and Rosenfeld in a series of 15,000 autopsies found 337 cases of septic meningitis. In over half the cases, the infection was secondary to middle ear or mastoid disease, while in only one-eighth was it due to disease of the accessory sinuses. We are apt to be led from the more common origin in the mastoid to forget the nasal sinuses as a portal of entry, and unfortunately the postmortem records have disclosed this situation in a number of instances. If we remember that middle ear infection is practically always preceded by infection in the nose or throat, we will not be so apt to forget the accessory sinuses as a vital factor. It is not uncommon, furthermore, to see a combination of middle ear infection with sinus disease, so that even in cases which present a frank infection of the middle ear and mastoid, we must not be misled into confining our investigation to that area to the exclusion of the nasal passages.

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The pathways through which infection has been shown to have invaded the meninges is known to you, but I believe that it is well to list them in the order of their importance and frequency: *1*. Blood vessels. *2*. Bony erosions. *3*. Perineural sheaths of olfactory nerve. *4*. Congenital defects in the walls. *5*. Preformed pathways.

It is logical to assume that a focus of infection which feeds through any of these routes should be eradicated, and the earlier it is removed the better. It is not my intention to describe methods of operating. These are standard procedures in which every otorhinologist is well versed. My conception of the operation is to produce wide open drainage of all areas adjacent to the dura, and by extensive exenteration of bony infection free the meninges from all connection with the factory of organisms which is feeding the toxins or bacteria into the brain. This may mean a simple mastoid operation with removal of the tegmen, or it may mean labyrinth drainage and removal of the petrous tip cells, or it may call for an extensive frontoethmoid sphenoid drainage. Whatever is necessary in each case must be done without hesitation and without delay.

I believe that every otorhinologist has meningitis in mind when he sees a severe infection of the ear and sinuses. Persistent headache is a warning which should never be overlooked. A stiffness of the muscles of the back of the neck is an important sign, just as rigidity of the rectus muscle is an important clue in appendicitis.

Photophobia in mild degree should not be passed over. Any or all of these signs in the presence of acute otitis media or acute sinusitis call for a lumbar puncture and they are definitely an indication for surgery. Especially is surgery indicated when the mastoid is sclerotic or poorly pneumatized. For in this type of bone the pathway of least resistance is by way of the meninges. Schelling has stated that the nonpneumatic mastoid constitutes an anatomic predisposition to meningitis. The blood and spinal fluid findings in early meningeal invasion are extremely important and will be dealt with by other contributors to this symposium.

Surgery of the ear and sinuses in early cases has no doubt added something to the therapy of meningitis, although pre-

vious to the advent of sulfanilamide, the prognosis of meningitis was practically hopeless.

J. G. Dwyer, of the New York Committee on Meningitis, has reviewed the collected statistics of meningitis and has placed the recoveries at less than 4 per cent. Kerrison, quoting Cunning in a paper not yet published, shows records as follows: New York Department of Health, 238 cases, 5 per cent recovered; A. A. Gray, 2,200 cases, 3 per cent recovered; Manhattan Eye and Ear, 2 per cent recovered; Johns Hopkins, no recoveries.

Since the use of sulfanilamide the record shows in a small series of cases: New York Department of Health, 11 cases, nine recoveries; Dr. Cunning, nine cases, six recoveries. In other words, in this series the recovery percentage was 75.

There is no evidence up to the present time that medical treatment alone will produce recovery in consecutive cases; therefore, it is essential that every possible bone focus be removed as soon as the diagnosis can be made. The clinical picture alone is not sufficient for specific information. We must know the phase which any given case presents in order to institute intelligent therapy. Spinal puncture repeated frequently and studied thoroughly furnishes the date upon which remedial measures are based. The blood studies also furnish important clues for the proper conduct of the treatment.

It is possible to say, therefore, in conclusion that we have a new confidence in approaching this problem. Everywhere more recoveries are being reported and the careful evaluation of our methods of treatment with the early institution of the measures at our disposal holds promise for a brighter future.

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1002 Carew Tower.

PHARYNGOMAXILLARY FOSSA INFECTION. CASE REPORT.*

DR. RAYMOND J. GAFFNEY, New York.

In presenting this case, nothing startling will be heard, but added stimulus will be given, I hope, to the study of neck infection. A few outstanding symptoms will be correlated with the anatomy of the region in question.

Historically, Bichat, in 1801, was perhaps the first to attempt the study of the compartments of the neck but, seemingly, accomplished nothing. A lapse to the later part of the last century was noted before accurate investigations into the pathways of infection from the neck to the mediastinum appeared in the literature. Only of late has renewed interest been noticed in this country. Furstenberg, Mosher, Iglauer, Coller, Yglesias and Pearse are some of the outstanding names in the literature. One year ago today, Dr. Loré read before this group his findings in neck infections of the planes, by dye injections, and by three-plane X-ray pictures. Even at the present time much can be done to clarify the proper mode of treatment and the description of the fascial planes, for we note through the literature marked divergence of opinions.

The opinion of the one group is bold surgery; that of another is to wait upon Nature's whims. The former opinion, I believe, is held by most. The general surgeon is free to admit that these infections have been badly handled, and further knowledge of the pathways of infection is in order.

Of course, figures speak out strongly, for when we read of New and Erich, of the Mayo Clinic, having treated 267 infections of the neck without one showing evidence of spread to the mediastinum, and with only eight deaths, we are forced to listen to their words, and those of Havens: pus does not burrow along the fascial planes as frequently as the anatomists would have us believe; pus is not an inert fluid, like water flowing from one compartment to another, but is an

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active material which whips Nature into real defense reactions. The result is walling off and pointing from enzyme digestion of tissues.

Mosher has called the carotid sheath the "Lincoln Highway" of the neck, but evidently all of Erich and New's cases stayed away from the highway, content to take only the local roads near home! Quoting Beck, however, "drainage placed in advance of involvement is better than that which only trails the involvement," we must accept as good surgical judgment. It takes much courage to stand by and see these cases each day become more toxic and worse locally, and not interfere surgically when we know that the carotid sheath can be invaded at any moment; for this sheath can be considered the posterior compartment of the pharyngomaxillary fossa. Such was the picture in the case presented this evening. At first glance, what enters our minds when we see a marked swelling around and above the angle of the jaw, and difficulty in opening the mouth, and the patient's complaint that he cannot swallow? Quinsy is a snap diagnosis, or dental pathology may be considered. We could outrule retropharyngeal abscess as belonging to the diseases of childhood as we are dealing with an adult male, age 46 years. Acute parotitis certainly must be thought of. We note that he has a temperature of 101° and appears very ill. His history tells us that he had quinsy 10 years ago, but nothing since. His dental condition is deplorable but nothing acute. His present history further states that he had pain, swelling and tenderness at the angle of the left jaw five days before admission to the hospital, with progressive dysphagia.

Upon inspection, as he lay in bed, his left face was badly swollen from zygoma to lower jaw, very tender over the parotid region and with no sign of fluctuation. His mouth was opened with great difficulty. That is a very definite symptom; namely, trismus, which is caused by splinting of the internal pterygoid muscle, which muscle enters into the formation of the outer and anterior boundary of the pharyngomaxillary space. That symptom alone should help us to locate the site of the trouble.

The etiology in this case is not clear. With the diagnosis of pharyngomaxillary abscess made, we admit that about 50 per cent of neck infections occur in this region following

trauma or as a result of tonsillectomy under local anesthesia with the piercing of the superior constrictor muscle by the needle. This muscle forms part of the inner wall of the space in question. Nothing of this sort occurred. Infected teeth have been mentioned in the history, but in most cases of infection of the neck from that cause, the symbiotic organisms of Vincent are found. This case showed a nonhemolytic streptococcus. We can give credence to the idea that the infection of the tonsil 10 years ago was the factor, but we don't know. Any tissue reaction, no matter how slight, sufficient to be reflected to a cervical lymph node, may cause a neck infection. Clinically, at least, we outruled the sinuses and the ear as the cause. To this point, namely, six days ago, there was fever but no chills, with a white blood count of 26,000. Sulfanilamide in large doses was administered at once. The patient was tided over for 24 hours, but the next day his discomfort and his local symptoms were worse.

The next important symptom is what we find in the throat. The uvula showed a mild glistening edema due to lymph stasis. The entire lateral pharyngeal wall was displaced toward the midline, with little or no change in color. Pressure in the area of the tonsil was not especially painful, and the tonsil appeared the same size as its mate. Peritonsillar abscess was promptly ruled out. But the pressure from without was sufficient to displace the inner wall structures; namely, the buccopharyngeal fascia, the superior constrictor muscle, the pharyngeal aponeurosis and mucous membrane, enough to show asymmetry of the two sides of the tonsillar region. The tongue was normal and the floor of the mouth negative, thus ruling out Ludwig's infection.

The swelling of the face was the one symptom evident on casual inspection. When we consider the close relation between the parotid gland and the pharyngomaxillary space, we note that the layer of deep fascia on the inner aspect of the gland is very thin compared to the outer covering, and very often deficient superiorly. It is readily seen that infection could spread from within outward to the parotid. I infer that this was the condition in our present case, but the subsequent course would make us believe that, if involved, it did not go to suppuration. No pus was evident at the mouth of Stenson's duct on pressure. Surgery, we believed, was

indicated in this case. Our courage was not sufficient to rely too much on Nature.

Twenty-four hours after admission, the patient was operated upon. We know that most of the cases reported in the literature followed local tonsillectomy, and that a longer time was allowed before operation, but this patient was acutely ill. Under local infiltration of novocaine the incision began half-way between the mastoid tip and the angle of the jaw, and extended well beyond the submaxillary gland toward the midline, just below the lower border of the mandible. As we know, the structures encountered were skin, superficial fascia, platysma muscle and the superficial layer of the deep cervical fascia. When we arrive at the latter structure, we usually recognize it as a fairly tough, resisting barrier. We incise this layer and then lay aside the knife. The facial vein, which was large, was not ligated but pushed aside. The submaxillary gland was well exposed and found to be perfectly normal. Recalling that the pharyngomaxillary space is internal to the parotid gland, and posterior and deep from above down to the submaxillary gland, we knew that we were approaching the area. By blunt dissection with the finger we followed upward just at the angle of the jaw until the styloid process was recognized. This must bring us to the posterior limit of the space in question, as the styloid process and the stylopharyngeal fascia form the posterior boundaries of the space, and the anterior boundary of the great-vessel sheath. It can here be appreciated how close we are to the "Lincoln Highway" of the neck. By a gentle sweep of the tip of the finger, all boundaries of the space were embraced. Upon withdrawal of the finger, we were rewarded by obtaining a small amount of malodorous pus, which was sufficient to establish our diagnosis. We used a Cirus drain pushed high up and closed the main portion of the incision by horsehair.

The after-treatment was uneventful except that for a few days postoperatively the patient had pain which required medication. Before he left the hospital his dental condition was improved by a few extractions. His total stay was just over two weeks.

The alternative treatment would be to delay from day to day, applying hot packs and waiting until the condition "pointed."

The average time in New and Erich's series was three weeks between onset and fluctuation. Just what the outcome in this case would have been at the end of three weeks I do not care to predict. Where would the pus have pointed? It could eventually have penetrated the tough fascia between the parotid and submaxillary glands, and it could also have gone down along the sheath of the great vessels into the mediastinum. If the pus were high enough it could eventually have eroded through the superior constrictor and pointed into the pharynx; but if it were not high enough and the incision was made, then we would be confronted with pocketing. This pocketing would defeat the whole object of the operation and external incision would be necessary.

To sum up: We have a patient with an acute involvement of the deep fascial compartment of the neck; namely, the pharyngomaxillary. The cause is unknown, but was perhaps of dental origin. The onset was very acute and the systemic reaction severe. After six days the decision to adopt surgery was adhered to, and the patient made a fairly rapid recovery.

A few lantern slides will now supplement the brief correlation of the pharyngomaxillary boundaries and the symptoms given in the text.

By this very brief presentation it is hoped that renewed interest in the anatomy of these parts will be awakened.

15 W. Eleventh Street.

OSTEOGENIC FIBROSARCOMA OF THE TEMPORAL BONE.*

DR. HAROLD W. CORYA, New York.

The patient was a married man, age 59 years, who was admitted on Aug. 21, 1939, and died Aug. 31, 1939. I was asked to see him one week previous to his admission, at which time his chief complaint was a constant excruciating pain over the left mastoid region. Examination at his home revealed an emaciated individual with a complete facial paralysis of the left side and a paralysis of the left vocal cord. Persistent questioning revealed that the patient had had a discharging left ear in childhood which lasted for some time. He had had no other trouble with the ear until his present illness.

The past history was furnished by Dr. William Seaman Bainbridge, to whom the patient was referred in May, 1930, at 50 years of age, for probable cancer of the stomach and throat. Careful examination revealed lues, and under systematic antisyphilitic treatment all symptoms disappeared. For seven years he had periods of treatment, but in 1937 of his own volition he discontinued all treatment.

In January, 1939, he returned to Dr. Bainbridge. Examination revealed moderate elongation of the arch of the aorta, paralysis of the left vocal cord and subacute left supraorbital neuritis. The throat and chest were negative on X-ray examination with the exception of the left vocal cord. There was recurrent paralysis of the left nerve abductor. Bronchoscopy and esophagoscopy revealed recurrent laryngeal nerve paralysis on the left and sensitory disturbances of the superior laryngeal nerve on that side in the upper esophagus. He had increased difficulty in swallowing and speaking. It was agreed by all examining doctors that the condition was luetic, and antileutic treatment was instituted.

For two weeks the patient required tube feeding. He improved under treatment so that the pain on the top of the

*Read by invitation at the New York Academy of Medicine, Section on Otolaryngology, Dec. 20, 1939.

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head and in the back of the neck, which had come on with the inability to swallow, gradually disappeared. His speech improved and he was able to swallow solid food. He gained in weight and was up and about. The headache disappeared. There was no tenderness over the mastoid at any time.

In June, 1939, when he evidenced the first sign of trouble with his left ear, some granulations appeared in the external auditory canal near the drum, where the patient had been inserting various things and had caused irritation. He also developed a facial paralysis on the left side, which gradually became complete. Specimens of granulation tissue sent to different laboratories brought four diagnoses: negative, luetic, carcinomatous and sarcomatous.

Examination of the ears revealed the right ear to be normal, and the left canal was completely filled with a mass of tissue, very hemorrhagic to touch. A piece of this mass was removed for biopsy and was reported by Dr. Eggston to be a sarcoma of fibro-osteogenic type. A left radical operation was advised at this time, and the patient was admitted to the hospital. He was almost totally deaf in the left ear.

Blood examination on admission showed hemoglobin, 63 per cent; red corpuscle, 3,250,000; leukocytes, 12,000; polys., 81 per cent. Complete blood chemistry was essentially normal.

The X-ray report was as follows: The left mastoid is large and cellular. There is a great deal of bone absorption involving practically all the mastoid cells, as well as the petrous ridge. The appearances are not those of ordinary inflammatory absorption but more like that produced by new growth. Tip region: There is absorption involving the cortex. Some remaining cells are seen superior. The right mastoid is small, cellular and clear.

The blood Wassermann and Kahn tests were negative. The urine showed faint traces of albumin.

A left radical mastoid operation was performed two days after admission to the hospital.

OPERATION.

Under general anesthesia the usual mastoid incision was made. The upper one-third of the mastoid was sclerotic, with

very few cells present. The bone around the antrum was soft. The lower two-thirds of the mastoid was filled with a cauliflower-like mass of dull reddish tissue. The dural plate was completely gone and it was impossible to differentiate the lateral sinus from the dura. A large section of the posterior canal wall was loose and easily lifted out of the wound. The usual radical flap was made and the wound closed completely.

The patient's temperature remained normal for 48 hours and he then developed a chill, followed by a rise in temperature, with a subsequent intestinal obstruction. A nasal tube was inserted into his small intestine and frequent lavages of his intestinal tract were done. Feeding was done through this tube and glucose was given intravenously. The patient expired on Aug. 31, 1939, eight days after the operation. We were fortunate to obtain an autopsy.

AUTOPSY REPORT.

S. S., Aug. 31, 1939. The body is that of an emaciated white man, age 58 years. There is a mastoidectomy wound on the left side, which shows evidence of healing and no pus. On removing the skull, the dura is adherent to the skull. The surface of the brain is engorged. There are several small, grayish-white fibrous plaques along the superior border near the longitudinal fissure. The dura is adherent in the region of the left petrous pyramid and the basilar nerves. The cerebellum, pons and medulla were crowded over to the right side and downward. The cerebellum is adherent to the posterior surface of the left petrous bone.

On section, there is a small extension of the tumor into the superior anterior border of the cerebellum, forming a growth 1 x 0.5 cm.

The left lateral sinus is occluded and shows clot. The lateral ventricles show nothing unusual. The choroid plexus are normal.

Gross external examination of the base of the skull shows some erosion of the petrous pyramid of the left side. No pus nor distinct tumor tissue noted. This bone is soft and mobile. The posterior surface around the internal auditory meatus is covered with tumor tissue, 3 x 2.5 cm. The lower

part and basilar surface of the petrous bone is entirely infiltrated by tumor tissue, with very little bony substance remaining. The petrous pyramid is soft and friable, and easily detached from the remaining portion of the skull.

The tumor growth extends into the tissues of the neck around the blood vessels and nerves. The ascending part of the internal carotid appears compressed. All the tissues and veins in this region are matted together. The tumor surrounds the internal carotid artery and jugular vein for a distance of 6 cm. in the neck, and extends inward to the lateral wall of the pharynx.

The right pleural cavity shows adhesions and there are a few adhesions on the posterior wall of the left side. The lungs show areas of increased density, the pleura is thickened in areas. The lungs fail to show distinct metastatic growth but show some areas of congestion.

The spleen is not remarkable.

The liver shows some fat. The lobules are small, apparently atrophic.

There are a few fibrinous plaques in the pericardium, also a small amount of fat. The coronary vessels are sclerotic. The right heart is dilated. All the valves appear normal with the exception of the aortic, which shows some sclerotic and fatty plaques. The mouth of the left is surrounded by plaques. No distinct aortitis.

The pancreas is apparently normal except for a small cyst in the tail, which feels indurated generally.

Kidneys: The capsules strip easily. The kidneys are somewhat swollen and show a few small sclerotic plaques. Markings are normal. The cortex of both kidneys shows some pitting from scars; arteriosclerotic type. Considerable sclerosis of right kidney. Thoracic and abdominal aorta show sclerosis and a few plaques. Right ureter shows considerable dilatation. Adrenals appear normal.

The mesentery is free of growth or any enlarged glands.

There is an area of induration and erosion of the stomach near the pyloric antrum, and also along the greater curvature of the stomach area. The rugae are thickened and enlarged

over this area, which measures 15 x 4 cm. The mucous membrane looks atrophic and eroded.

The duodenum shows areas of atrophic mucosa but no distinct erosion. There is some engorgement which extends into the jejunum. No distinct ulceration found. Jejunum and ileum show slight engorgement. The colon is negative in its entire length.

Microscope slide of tumor tissue in the neck shows numerous bizarre-shaped spindle and round cells, some being large giant cells, some medium and small spindle cells. Most of the cells show atypical mitosis, large vesicular nuclei with nucleoli. There is considerable vascularity; also large areas of necrosis. There is considerable fibrous tissue matrix which contains considerable hyalin; in areas, considerable fibrilla.

The section taken in the region of the base of the petrous pyramid shows practically complete loss of bone, having been replaced by tumor tissue composed of cells similar to those in the neck. The cells run in various directions, forming strands and whirls, but form no definite structure. Many of the cells are multinucleated giant cells and show an abundance of cytoplasm.

In some sections of the tumor, the cells appear not unlike epithelial cells, and infiltrate the adventitia of the larger vessels. Some of the blood vessels are thrombotic.

Microscope slide of cerebellum shows invasion by tumor cells similar to those in the petrous bone, with complete replacement of the nervous structures of the cerebellar tissue by the tumor cells. Microscope slide of brain shows some fibrosis of the pia mater in areas, with hyalinization. The cortex is edematous.

Microscope slides of heart show albuminous degeneration, fragmentation, muscle. The muscles are hypertrophied and show increase in fibrous tissue. There is arterial fibrosis. Microscope slides of aorta show hyalin fibrous thickening of the intima, with some areas of cholesterin, slight calcification, some fragmentation elastic lamina. There is no inflammatory reaction.

Microscope slides of adrenals show some fat, albuminous degeneration in the medullary region and in the zona reticulata; some lymphocyte reaction.

Microscope slide of lung tissues shows many alveoli filled with pus, edema, some congestion and hemorrhage. Some bronchi contain purulent exudate. In areas, the exudate is so extensive as to form abscess.

Microscope slide of spleen shows fibrosis, congestion and hemorrhage.

Microscope slide of intestine shows some erosion of the mucosa and congestion.

Microscope slides of pancreas show nothing remarkable with the exception of an encapsulated nodular mass, which has a capsule and shows a papillary hyperplasia of cells that are more of the isle of Langerhan's type.

Microscope slide of bronchial lymph node shows anthracosis, congestion, edema, some lymphocytic hyperplasia.

Microscope slide of kidney tissue shows marked tubular degeneration. The glomeruli are swollen and congested. There are some areas of lymphocytic inflammatory reaction, some areas of polymorphonuclears in which appear some bacterial colonies.

There is considerable arteriosclerosis. Many tubules contain numerous casts in which are some granules.

Microscope slides of liver show albuminous degeneration, some congestion, some inflammatory reaction around the bile duct.

Anatomical Diagnosis: Osteogenic fibrosarcoma of the temporal bone, probably petrous pyramid, with extension into the neck and cerebellum. No distant metastasis. Myocardial degeneration. Bronchial pneumonia. Small adenoma of pancreas, isles type.—A. A. Eggston, Pathologist.

30 East 40th Street.

MENINGITIS AND TEMPORAL LOBE ABSCESS SECONDARY TO SUPPURATED PETROSITIS. CLINICAL COURSE WITH SULFANILAMIDE.*

DR. EUGENE R. SNYDER, New York.

This is a case report of a female, age 23 years, who was admitted to the Mount Sinai Hospital on March 22, 1938, with a four-day history of pain in her left ear following an acute upper respiratory infection, and in 48 hours complained of dizziness and falling to her left.

Upon examination the temperature was found to be 103° and there was evidence of an acute suppurative process in the middle ear. Neurologically, the only abnormal finding was that the patient walked with a wide base. Twenty-four hours after admission she developed a marked horizontal nystagmus upon left lateral gaze. The fundi were normal.

She was observed very closely on the ward. The otitic process progressed so that on the thirteenth day of her illness a simple mastoidectomy had to be performed. The mastoid cells were found to be full of pus, though there was no destruction of intercellular substance. The culture of this pus demonstrated the hemolytic streptococcus. The temperature, as stated, on admission was 103°, and then fictuated between 99,° 101° and 102° in the hospital. Thirty-six hours after operation it rose to 104,° and a blood culture was then taken. This and subsequent cultures were found to be sterile.

Thirteen days after operation the patient began to complain of severe left retro-orbital pain, the discharge from the middle ear and the mastoid fistula became profuse. Because of the persistence of pain and the profuse discharge for 10 days, surgical investigation of the petrous pyramid was deemed advisable.

A preliminary spinal tap was performed to diminish the amount of intracranial tension and facilitate exposure of the

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roof of the petrosa. After removal of a large portion of the squama and as the temporal lobe was elevated from the superior surface of the pyramid, a gush of thick, creamy, nonodorous pus poured out and emptied itself from a pit about 1.5 cm. beyond the arcuate eminence. A rubber dam drain was inserted between the dura and the petrous pyramid. Culture of this pus also revealed hemolytic streptococcus.

Forty-eight hours later, the patient became drowsy, responded to questions and commands slowly, and there was a horizontal nystagmus to both lateral directions. There was some nuchal spasm but no Kernig or Babinski. A spinal tap was performed and the fluid was found to be cloudy, Pandy 3+, but no organisms were found on smear. Four days later, the temperature rose to 106.2° and the patient developed definite meningeal signs. The cerebrospinal fluid that day was found to be cloudy, the cell count was 4,500 cells per cm.; the next day, 11,000 cells, and this upon culture was reported positive for streptococcus hemolyticus. Fundus examination now showed minute hemorrhages scattered on the deeper and superficial layers of the retina, with blurring of the upper disc margins.

With the onset of meningeal signs, sulfanilamide therapy was instituted. She was given 25 gr. every four hours, which was later reduced to 15 gr. The concentration in the blood was kept at levels between 8 and 10 mgm. per 100 cc. The drug was continued for nine weeks. Spinal taps were performed daily from April 25 until July 6, when the fluid became clear and free of organisms.

During this time the signs of meningitis abated slowly but clinical evidences of a brain abscess now appeared. The left nasolabial fold became shallow, she was unable to name objects and there was progressive blurring of the right disc.

The wound was revised and again pus was found exuding from the original site of the petrous pyramid beyond the arcuate eminence. One week later, the patient developed a distinct anomia, began vomiting and there was further blurring of both optic discs. A right mimetic facial paresis now appeared and also a right Babinski.

Exploration for possible brain abscess of the left temporosphenoidal lobe was advised and performed. No abscess was

encountered but a small portion of the brain tissue sent to the laboratory was reported as degenerated brain substance, probably from an inflammatory process.

Shortly following the above procedure the patient commenced to show psychic changes; that is, irritability, crying, inability to name objects, etc. These episodes disappeared, however, and the patient's general condition rapidly improved so that 12 weeks after the onset of her illness the middle ear was dry, the mastoid wound healed, and she was entirely symptom-free. The only abnormal sign was persistence of blurring of both fundi. The patient was discharged from the ward service four months after admission to our out-patient department.

She continued to show signs of improvement, enabling her to return to her occupation as a waitress. She was seen in the neurological follow-up clinic on Dec. 6, or five months after her discharge from the hospital, and the only abnormal sign at that time was a slight speech defect.

Six days later she developed what she said was a severe head cold, and four days later complained of pain in her left ear. She then noticed that there was a swelling behind this ear. She was readmitted to the hospital, where the post-auricular abscess was incised and drained, and again sulfanilamide therapy was administered. Shortly afterwards, she complained of severe left parietal headache, developed projectile vomiting and became drowsy. Examination showed a fairly marked acute pansinusitis, a right homonymous hemianopsia and a pulse of 60 per minute. The patient had difficulty in naming objects, the fundi showed blurring of both discs and the hearing in the left ear was slightly impaired. Ventriculograms demonstrated dilation of the right lateral and the third ventricles, with displacement to the right.

Exploration of the temporal lobe by the neurosurgeon was again performed. A firm mass which gripped the needle was encountered in the posterior portion of the temporal lobe but no pus was obtained.

The subsequent postoperative course was an exceedingly stormy one. Right-sided hemiplegia developed, vomiting became intense and the papilloedema increased. The impres-

sion was that a very thick abscess capsule was encountered and that a subtemporal decompression should be performed until the patient's general condition improved; however, the patient suddenly collapsed, she became very cyanotic, the pulse was thready and she was then placed in a respirator.

Because of the rapid onset of coma, a large ventricle needle was inserted through the old scar, and at a depth of 3.5 cm. about 15 cc. of thick pus escaped. The condition of the patient became rapidly worse and she ceased breathing shortly thereafter.

POSTMORTEM REPORT.

1. Multilocular left temporosphenoidal brain abscess with an exceedingly thick capsule.

2. Encephalomalacia.

3. Recurrent mastoiditis. There was no evidence of meningitis.

SUMMARY.

1. The clinical course of a streptococcic meningitis under the influence of sulfanilamide is reported.

2. The surgical and chemotherapeutic measures employed resulted in recovery from an acute suppurative meningitis.

3. Fatal outcome was due to a flare-up of a latent temporosphenoidal brain abscess.

1100 Park Avenue.

COMBINED MEETING OF THE NEW YORK ACADEMY OF MEDICINE AND THE PHILADELPHIA COLLEGE OF PHYSICIANS.

SECTIONS ON OTOLARYNGOLOGY.

March 20, 1940.

Introduction: Dr. Jacob L. Maybaum.

This is indeed a unique occasion, for we meet here tonight to discuss an epochal advance in otology. During the last three years definite progress has been made in stimulating our interest in the subject of surgery for otosclerosis. We all know that credit must be given to the pioneer workers in this field, Barany and Holmgren, and more recently, Sourdille. Dr. Lempert deserves special credit for his outstanding contribution for two reasons: (1) he has perfected an operative procedure for otosclerosis so perfect from an anatomic-surgical viewpoint as to defy, thus far, any attempts at modification. This is a one-stage operative procedure, in contradistinction to those used by other operators; (2) the results he has obtained are far superior to those of any previous operator.

Tonight, Dr. Lempert will discuss his experience in all its phases, stressing particularly the end-results obtained in 150 cases upon whom he had operated during the past three years. He will be followed by four discussers who will take into consideration an additional total of 100 cases operated on by them by the Lempert procedure. The speaker does not need an introduction. He has been invited and has appeared at many Otological Sections throughout the country and has succeeded in focusing our attention upon a very important fact: that by means of well directed surgery, permanent improvement in hearing can be obtained in many well-selected cases of otosclerosis. This evening Dr. Lempert will discuss "Endaural Fenestration of the Horizontal Semicircular Canal for Otosclerosis. Indications, Technique and Observations as to Early and Late Postoperative Results." I take great pleasure in introducing Dr. Julius Lempert.

Endaural Fenestration of the Horizontal Semicircular Canal for Otosclerosis. Indications, Technique, Observations as to Early and Late Postoperative Results. Dr. Julius Lempert (by invitation).

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. JACOB L. MAYBAUM: Those of us who have worked with Dr. Lempert during the past year and have in addition observed his operative skill soon came to the conclusion that in order to approach his perfection of technique and end-results, it is necessary to attend to the minutest details of the procedure employed by him. Any attempt to modify the technique only results in added difficulties and problems. It will be interesting, indeed, to have a group of men who are doing this work today report a year hence regarding their results. I can recall that hardly more than 13 months ago the average operating time of Dr. Lempert for his otosclerosis cases was four or five hours. Today, he seldom requires more than two hours, and with that has followed an improvement in the final results, such as he demonstrated for us this evening by means of some of his charts. Such should be the goal of those who wish to take this work seriously.

Dr. Lempert will demonstrate some of his end-results in the anteroom soon after the close of this meeting. I am sure I voice the opinion of the members of this combined meeting that this has been a memorable occasion. I express, I am certain, the opinions of the members of both Sections in extending our thanks and appreciation to the reader of the paper and to those who have been kind enough to come long distances to discuss his paper.

DR. EDWARD H. CAMPBELL: My discussion on this subject of labyrinth fenestration for deafness will be based largely on my personal experience with the operation, rather than on the material presented this evening by Dr. Lempert. I shall discuss to some extent the results I have obtained in the performance of 22 operations done according to the Lempert technique and two revisions. I shall express some views based on this experience that are in accord with Dr. Lempert's views and shall express others that will conflict with his ideas.

At the present time, there is considerable skepticism in the minds of many otologists as to the real value of this work, particularly the permanency of the good results claimed and the necessity for the elaborate and intricate operative technique. In order to overcome this skepticism and for this work to gain the approval of those otologists whose favorable opinion is so essential for fullest advancement of this work, it is necessary that the good results reported by Dr. Lempert be confirmed by other otologic surgeons. The results reported at this discussion tonight should do much to either encourage or discourage further efforts along this line, for by this time other otologic surgeons will have done a sufficient number of these operations to have produced results of one type or another and will have developed opinions based upon at least a moderate amount of experience.

We have learned through the experience of Holmgren, Sourdille and other pioneers in this field of work that fenestration of the labyrinth, particularly a semicircular canal, will produce an immediate improvement of hearing in suitably selected cases. We have further learned through Dr. Lempert's technique that the labyrinthine fenestra can be made to stay open. This is undoubtedly proved by an examination of Lempert's cases, many of whom have been operated for more than two years and still give an active fistula response, and by some of the cases of others of us who have operated cases a year or more ago. These cases that retain the active fistula reaction undoubtedly retain a great deal of the improvement of hearing that was gained by the operation. In my own cases that have now been operated sufficiently long to be judged, I have found that the degree of hearing retained is directly proportional to the activity of the fistula response. That is, in those cases of mine over a year old in whom the fistula reaction is fairly active there has been a retention of much of the improvement of hearing that was present immediately after operation. On the other hand, in those cases in which the fistula response has gradually diminished, there has been a proportionate decrease in the hearing. In none of my cases that are a year or more old have I been able to retain the greatest amount of hearing gained after the operation, but this is consistent with the fact that in none of my cases has the fistula response remained as active a year from the operation as it was during the first four to six weeks. Is this the result that is to be expected from this operation; that is, the retention of a certain amount of the hearing gained by operation, or will an exactly perfect technique result in the retention of the full amount of hearing gained by the operation with presence of a very active fistula reaction?

The retention of a moderate improvement of hearing but not the greatest that was obtained after operation is illustrated in four of my cases. I shall express their hearing loss in percentages which is based on the 512, 1,024 and 2,048 frequencies.

The first of these cases was operated upon 17 months ago. Before operation there was 61.6 per cent loss of hearing in the operated ear. Ten days after operation the hearing had improved to 31.7 per cent loss, a gain of

30 per cent. During the subsequent few months there was a gradual loss of this improvement until at the end of a year following operation there was 42.7 per cent loss of hearing, a gain of 19 per cent over the hearing before operation. There has been no further loss since then and the fistula is fairly active (after 17 months), but not as active as during the first month or two.

The second case of this kind was operated upon 14 months ago. The hearing before operation was 62.4 per cent loss in the operated ear. The most improvement was present six weeks after operation when there was 25.3 per cent loss, an improvement over the preoperative hearing of 37 per cent. During the next few months there was a gradual loss of this improvement until at the end of eight months from the operation the hearing was 41.3 per cent loss, an improvement of 21 per cent over the preoperative hearing. The fistula is moderately active but not as active as when the hearing improvement was at its best.

A third case was operated upon 10 months ago. Before operation the hearing loss in the operated ear was 37.3 per cent. The best improvement was present one month after operation when the hearing loss was 12 per cent, a gain of 25 per cent over the preoperative hearing. During the following few months there was a gradual loss of some of this improvement and at the end of seven months from operation the hearing loss was 21.3 per cent, a gain of only 16 per cent over the preoperative hearing. In this case also the fistula reaction is fair but not as good as one month after operation.

A fourth case was operated six and one-half months ago. Before operation the hearing loss was 42.7 per cent. Three weeks after operation the loss was 13.3 per cent, an improvement of 29.5 per cent over the preoperative hearing. Subsequently there was a gradual loss of some of this improvement, and at five months after operation the loss was 33.3 per cent, an improvement of only 9.5 per cent over the preoperative level. There is still present a fairly active fistula but not nearly as active as when the hearing was at its best.

These four cases illustrate the considerable improvement of hearing that may be obtained shortly after operation with the gradual reduction of some of this improvement during the subsequent few months, but a retention of sufficient improvement to be of benefit to the patient. They further illustrate the reduced activity of the fistula response several months after operation over what it was when the hearing was at its best. This reduced fistula response I attribute to partial closure of the fistula, either by bony regeneration or fibrous tissue. I believe that such cases as these will represent many of the operated cases which we must classify as only partially successful. The permanent retention of a moderately active fistula response will give, therefore a moderate improvement of the hearing, but if only the amount of hearing is gained as represented by these cases it seems doubtful if such results are worth while. Such patients are not satisfied, largely because they have experienced a considerable improvement when the hearing was at its best a few weeks after operation, and with the subsequent partial loss they have been disappointed and depressed, believing the operation has been a failure, although as in these cases there has been an improvement of the hearing sufficient to have given them distinct benefit.

Since having had the advantage of a close examination of some of Dr. Lempert's patients, many of whom had been operated from one to two and one-half years and have retained an extremely active fistula, I have concluded that such cases as I have illustrated above have been only partially successful because of some fault in the operative technique which has allowed partial closure of the labyrinthine fistula. With a perfect technique, therefore, that would prevent any closure of the fistula, the greatest amount of hearing improvement should be retained. I believe the fault in the technique of these cases has been a failure to secure a sufficiently large fistula.

What would constitute a perfect technique so that such results are possible? Will it be the combination of the intricate surgical reconstruction of the

middle ear, the removal of the malleus head, etc., with the proper fenestration of the labyrinth, or is the secret of the success the proper handling of the labyrinthine fenestration, with perhaps no necessity for elaborate plastic surgery of the middle ear? That practical improvement can be obtained with faults in the reconstructive technique can be illustrated by several of my cases.

One patient who was operated upon 21 months ago has retained an improvement of 21.4 per cent of hearing in spite of the fact that the tympanomeatal skin flap was destroyed and it was necessary to dissect a skin flap from the anterior canal wall and swing it across the lumen of the auditory canal to close over the fenestrated external semicircular canal. The best hearing obtained after operation has been retained and the fistula response is as active as shortly after the operation.

In another patient operated upon nine months ago, the tympanomeatal flap was badly lacerated and the malleus head was not removed. Fragments of the skin flap were fitted over the fenestrated external semicircular canal. One month after operation the hearing had improved from 42.7 per cent loss to 20 per cent loss. During the following few months there has been a partial loss of this improvement coincident with a gradual reduction of the activity of the fistula. At present, nine months after operation there is only 8 per cent improvement of hearing, with the fistula only slightly active.

In another patient, operated one year ago, the malleus head was not removed. One month after operation the hearing had improved 20 per cent but during the subsequent months all improvement was lost coincident with gradual loss of the fistula reaction.

These three patients illustrate a practical improvement of hearing in spite of a faulty or incomplete technique, particularly in connection with the reconstruction of the middle ear. In two of the cases this improvement was only temporary but in the other, all improvement has been retained. The loss of the improvement in these two cases in which the malleus head was not removed I have attributed to the partial or complete closure of the fistula and not to the fact that the sulcus tympanicus was not removed nor the head of the malleus amputated. It appears logical to me to assume that if the hearing was improved 20 per cent or more for four to eight weeks after operation that this improvement would have been retained if this fistula had not closed.

Dr. Lempert has contended that the reconstructive technique is necessary so that the epithelial-lined surface of Shrapnell's membrane can be displaced to cover over the fistula which has much to do with the prevention of closure of the fistula by bony regeneration. He has also emphasized the importance of hermetically sealing the reconstructed widened tympanic cavity with the tympanomeatal skin flap in such a way as to enclose the fenestra in the external semicircular canal within the area of this newly created air space. How important this reconstructive technique is remains to be proven. The fact remains that in the absence of this part of the technique that practical improvement of the hearing can be obtained when the fistula is properly created. Whether or not it is going to remain necessary to stretch the drum so that the epithelialated surface of Shrapnell's membrane covers the fistula also remains doubtful, for the application of a Thiersch graft, as advocated by Lempert, may serve the same purpose of supplying an epithelial surface.

In regard to the indications and contraindications for this operation, I shall have to take a view contrary to Dr. Lempert's in one particular. He has stated that previous infection of the middle ear and a perforation of the drum should be contraindications for the operation. While it is logical to believe that the best results will be obtained in those typical cases of otosclerosis in which there is no history of middle ear infection, it does not seem necessary to me to exclude those cases from operation that give a history of middle ear infection or when there is a thickened, scarred and even perforated drum. If it could be proven in such cases that there was actually

some damage to the round window which would prevent it from functioning normally, then it would probably be necessary to eliminate such cases from operation.

To prove such a condition present does not appear practical and to assume that all middle ear infections have left a poorly functioning round window is not reasonable.

In my series of cases I have not felt that a history of middle ear infection at one time or a thickened and scarred drum were contraindications, if the factors of bone conduction, vestibular functions, etc., were favorable. My series includes six such cases where the history of abscesses (usually none since childhood) and the presence of scarred drums with healed perforations were present. In all of these cases there has been a satisfactory improvement of hearing following the operation. In one of these cases there was found an ankylosis of the incudomalleolar joint. Even with considerable effort it was not possible to separate these two bones. In two of these six cases the early improvement of hearing following operation was lost within three months, due to closure of the fistulae. In three of the cases there has been some loss of the early improvement of hearing following operation coincident with partial loss of the fistula response. In the sixth case, performed six weeks ago, the hearing improvement has been considerable. These cases impress me with the fact again that in spite of the abnormal drum and possibly middle ear cavity from previous infection, improvement can be obtained by fenestration of the labyrinth, and the indications point to a retention of this improvement if the fistula remains open.

A brief summary of my cases will reveal the following results:

Three cases operated, respectively, 21, 17 and 14 months ago have retained hearing improvement of 21.4, 19 and 20.5 per cent with a fairly active fistula now present. Another patient shows an improvement of 21 per cent six months after operation.

Three cases show slight improvement as follows:

One case an improvement of 8 per cent nine months after operation, with slightly active fistula.

One case an improvement of 9.5 per cent seven months after operation, with slightly active fistula.

One case an improvement of 9.5 per cent five months after operation.

Five patients showed a satisfactory improvement for a few months after operation but subsequently have lost all the improvement coincident with loss of the fistula reaction. Two of these cases were revised, with improvement which again lasted for less than two months.

Eight cases have not been improved at any time due to having been unsuitable for operation, bone conduction having been poor in all of them and a dead labyrinth in one.

The remaining two cases were operated five and six weeks ago and I believe will be the best of all my cases. The operative technique was carried out in every detail and the fistulae in the external semicircular canals were made much larger than any of the previous ones. At present both show a remarkable improvement of hearing with extremely active fistulae.

The experiences gained in the handling of these cases has caused me to form the following opinions regarding this operation:

(1) That a practical and permanent improvement of hearing can be obtained in suitably selected and operated cases.

(2) That by far the greatest factor for success is the proper creation of the fenestra in the labyrinth.

(3) That in many of the operated cases there will be some loss of the improvement obtained shortly after operation by a partial closure of the fistula by bony regeneration or fibrosis.

(4) That the reconstructive part of the operative technique may not be necessary for good results provided the fistula is preserved.

(5) That fair to good results may be obtained even if such errors of technique as laceration of the skin flap or drum occurs.

(6) That a previous middle ear infection or scarred drum are not contraindications provided other factors remain favorable.

(7) That, while in general, improvement of the tinnitus runs parallel with improvement of the hearing, there may be relief from the tinnitus without improvement of the hearing. This was illustrated in one of my cases.

(8) Hearing results cannot be considered final or permanent for at least one year after operation.

DR. GEORGE E. SHAMBAUGH, JR.: My discussion will be in three parts: (1) An evaluation of Dr. Lempert's results; (2) an evaluation of my own results with the Lempert operation for otosclerosis; (3) a discussion of a few of the many interesting problems and questions that have been raised by this new procedure.

For nearly two years I have followed Dr. Lempert's work by numerous visits to New York, by letters and telephone conversations, and by patients whom I have referred to him. Dr. Lempert's results speak for themselves. There has been the usual opposition to his work that accompanies any great advance in medicine. Much of this opposition has been based on honest skepticism. Suffice it to say that Dr. Lempert's patients are available for all to see and those who have taken the trouble to see the results no longer doubt that in Dr. Lempert's hands the patient with otosclerosis and without too much secondary nerve degeneration may expect to recover the major part of his hearing loss through an operative procedure devoid of risk to his life and with very little risk to his hearing (Slide 1).

In June, 1938, I spent several weeks with Dr. Lempert learning the technique of the operation on the cadaver. When he announced that I was ready to operate on a patient I returned home, found a patient willing to try this new procedure and we both survived five hours of the hardest, most exacting and exhausting work that I had ever let myself in for. Two weeks later the patient left the hospital with a very satisfactory gain in hearing averaging 17 decibels for the conversational frequencies (Slide 2). I was encouraged to continue the work and have now completed a total of 20 operations on 17 patients with otosclerosis.

For the first year encouraging initial results were soon followed by discouraging and disheartening end results (Slide 3). Nine patients were operated, two of them twice. As the chart shows, the initial hearing gain two to four weeks after operation was good in nearly all but the end result was negative in all but one patient (Slide 3a). To me the most discouraging thing was that in every operation the technique as devised by Lempert was faithfully and, as far as I could see, successfully followed, and yet most of the fistulas closed by new bone formation (Slide 3b). For awhile I did no more operations. Then continued observation of Dr. Lempert's work plus experimental work on bone regeneration by Dr. Fowler Jr., and others began to indicate the importance of bone dust particles in bone regeneration. I began to use extreme care to remove every particle of bone dust while making the fistula (Slide 4). Of nine patients operated from November, 1939, to the present date the fistula has closed only once. It seems likely that bone dust was the main factor in the closure of my early fistulas (Slides 5-8).

To facilitate the removal of every microscopic particle of bone dust I have devised a simple irrigating apparatus which fits on to the self-retaining retrac-

tor that I use (Slide 9). A small stream of warm normal saline flows into the cavity in the region of the incus through one tube and is sucked out by the other tube in the region of the solid angle. A thin film of saline thus flows constantly over the fistula during the use of the polishing burr carrying away each particle of bone dust as fast as it is created. On theoretical grounds and in practical use, I think that this may solve the problem of bone dust as a cause for bone regeneration. However, further experience by myself and others will be necessary before we will know the value of this addition to the technique. I have used this device in my last four operations and so far am very encouraged by the results (Slide 10-12).

So far the problem of closure of the fistula by new bone formation has been the greatest obstacle to success in otosclerosis surgery (Slide 13).

Second in importance as a cause for failure is postoperative serous labyrinthitis. In one way serous labyrinthitis may be more serious than bony closure because the closed fistula may be reopened but a labyrinth seriously damaged by inflammatory changes cannot be repaired. A mild degree of serous labyrinthitis occurs relatively frequently after the Lempert operation and is manifested by a temporary depression in hearing and in the more severe cases by a temporary suppression of the fistula test lasting from a few days to a week or two. The symptom of diplacusis, as I pointed out a year ago, is definite evidence of an inflammatory reaction in the cochlea with an edema or thickening of the vibrating membrane. About half of the patients will show a transient diplacusis the first week after the operation. Fortunately the severe cases of serous labyrinthitis are rare. In these cases the fistula test never returns and there may be a permanent impairment of cochlear function.

The larger the fistula the greater the hearing improvement, but also the greater the likelihood of a serous labyrinthitis. Since there is no effective treatment for postoperative serous labyrinthitis except to remove the packing early, prevention is our chief weapon and prevention is best affected by keeping operative trauma and inflammatory reaction in the wound to an absolute minimum. The endaural approach has an important advantage over the postaural in this respect. The maintenance of the strictest surgical asepsis during the operation and in the entire postoperative care until complete healing has occurred is an absolute requisite. Finally, perfection and finesse in technique will minimize the operative trauma to the tissues and particularly to the membranous labyrinth.

Critics of the Lempert operation state that the patient who can be benefited by the operation will receive equal or greater benefit from a properly fitted hearing aid without the hazards of surgery. This is not so. My first patient who required a total of three operations over a year and a half before his hearing gain was maintained used one of the better aids before the first operation and again when the first and second fistulas closed. In his own words, "there is absolutely no comparison between the improvement experienced from the operation and the help given by the hearing aid". First, the degree of hearing improvement is much greater after the operation. Second, the quality of the sounds heard is now entirely natural and with the hearing aid voices were always metallic and artificial. Third, the hearing aid caused confusion due to the uneven amplification of sounds and noises. And fourth, with the hearing aid he could never tell which direction the sound was coming from and now he has no difficulty in locating the speaker. This patient was then asked frankly whether the end result justified, in his estimation, the three separate operations, and there was not the slightest hesitation in his answer. Incidentally, a salesman of one of the wellknown hearing aid makers has taken the pains to describe to this patient the grave hazards to life from the Lempert operation with the statement that even should he survive the operation his hearing improvement would be too slight to avoid a hearing aid and would be too brief to be worth while. The campaign being carried on against the Lempert operation by this firm is sufficient proof that otosclerosis surgery has a future.

With regard to the mechanism of the improved hearing after the Lempert operation, I believe that the transmission of sound vibrations from the drum membrane to the fistula is essential for the best results. For this mechanism to function the incus must remain in situ with an intact blood supply, it must be mobilized by severance of the incudomalleolar joint and removal of the head of the malleus and the connection between Shrapnell's membrane and the pars tensa must remain intact. So far I have not had the misfortune to dislocate the incus or to tear the attachment of Shrapnell's membrane so that I speak from theory and from observation of the work of others rather than from experience. I am not certain that Shrapnell's membrane ever entirely covers the fistula and in some cases I doubt that it reaches the fistula. However, the adjacent skin of the superior canal wall is equally thin and elastic and is equally capable of transmitting the sound vibrations from the drum membrane to the fistula.

In conclusion, I should like to state that those of us who are attempting to follow the footsteps of Dr. Lempert entirely owe what little success we have thus far attained to the hours of patient instruction by Dr. Lempert. This is an entirely new and different type of surgery requiring a special set of instruments, a special technique and special training. With special training and prolonged cadaver experience, good results are difficult enough to obtain. Without adequate preparation and training, the operation should not be attempted. The surgery of otosclerosis is still in its infancy, but already it is apparent that the Lempert operation marks a milestone in otology.

J. A. Sullivan, M. B.: May I first of all express to you, Mr. Chairman, my sincere appreciation and thanks for having invited me to participate in the discussion of this excellent presentation of Dr. Lempert's this evening.

It was my privilege to be present at the meeting of the Collegium in Groningen, Holland, July 1938, and to have listened to the paper of Professor Holmgren on "The Surgery of Otosclerosis" before that society. His paper was fully discussed by Professor Nager and Dr. Sourdille.

Since the advent in this country of Dr. Lempert's outstanding contribution before the American Otological Society in May, 1938, and the observations I made of Holmgren's, and Sourdille's results just previously mentioned, I, too, have pursued and carried on this problem in Canada.

Frankly, and I am going to be quite frank, this new epoch in Otology must not be judged by its failures but only by its successes. It is entirely a new field of otologic surgery where one mistake is invariably fatal not to be compensated for by nature which treats the otologic surgeon so kindly in disease of the temporal bone.

From September, 1938, to the end of February, 1940, I have carried out experimental procedures on monkeys and operated on 35 patients by the Lempert Technique. Of this number seven cases have retained their practical physiological improvement of hearing with open fistula for a period covering from one year to one year and four months; two cases for eleven months; two cases for eight months; two cases for six months. The remaining number comprise cases less than six months duration, failures, and revisions. I had the honour of having Professor Dohlman of Lund, Sweden, assist me in one of my operations. He stated—quotation "this procedure definitely offers the finest surgical effort to date and I believe (Dohlman) that, if coupled with an elucidation of the underlying cause which in addition must be treated, then we can say with some degree of finality that the solution to this heretofore baffling problem is at hand."

It is my opinion that, although audiograms register hearing loss and must be done in conjunction with routine hearing tests, they do not accurately record the patient's ability to understand the spoken word, this of course, being a factor which is also in part subject to the patient's intelligence.

Apparent reasons for failure: (1) Failing to meticulously perform each successive step in the operation—one mistake is invariably fatal. (2) Selection

of the improper type of case for surgery. (3) The exacting precision required in making the fistula coupled with the careful moulding and placement of the flap over the fistula. (4) Not recognizing damage to the membranous labyrinth at the time of fistulization.

Observations made: (1) Where a fistula has remained open for a period of two months or more and then gradually closes, I believe that case should be reoperated and the fistula reopened. I base this on three secondary operative cases of my own plus observations and reports of the work of Holmgren and Sourdille. (2) Where a fistula remains open for seven days and then closes off, allow the wound to heal thoroughly and fistulize again at a later date. (3) Where a fistula remains open for three weeks, accompanied by severe vertigo, then suddenly closes, the membranous labyrinth has been permanently damaged and reoperation is to no avail. (4) In conclusion: The tympanolabyrinthopexia is a surgical act which serves to adapt a new tympanic system to a permanent labyrinth window created on the osseous semicircular horizontal canal. (a) The intact tympanic membrane, considerably released and relaxed through the resection of the head of the malleus. (b) A second cicatricial membrane reposing on the incus, elongating the tympanic membrane as far as the labyrinth fistula covering the latter.

The fistula of the osseous canal is situated above its ampulla. Its permanency is difficult to obtain, but it is possible to achieve by different technical manipulations, (cognizant of Sourdille's and Holmgren's procedures, I most definitely prefer the Endaural approach of Lempert), and if necessary by one or even two reopenings, which is supposed to bring about a progressive weakening of the osteogenesis (as yet this has not been conclusively proved and must be accepted therefore with some degree of reservation).

Finally: (a) In otosclerosis cessation of the progress of the deafness can be obtained by the establishment of a permanent labyrinth-fistula. But to obtain an important lasting improvement of the hearing power, a second condition is required: the adaption to this fistula of a new tympanic system as amplifier of the aerial sound vibrations. (b) The duration of the functional benefit depends on the permanence of the anatomical modifications. This permanency is difficult to obtain and it is possible in a high percentage of the cases: this is in the first place a question of operation technique and of the patient to be operated on. (c) The principle of the effectiveness of the surgical treatment in otosclerosis, affection hitherto considered as incurable, seems undoubtedly established. But in practice there are serious difficulties, which obliges one to approach the problem with great precaution and after patient preparations.

Dr. William John Greenfield: In the discussion of Dr. Lempert's paper, my contribution will be most helpful by agreeing or disagreeing with known facts regarding the practicability of the endaural fenestration operation of the labyrinth for the permanent improvement of hearing. My observations are based on the experiences with eighteen patients whom I operated according to Dr. Lempert's special technique described in his original article. The first seven cases were operated at Dr. Lempert's surgical clinic under his helpful guidance. The next three cases were operated at the Hackensack Hospital. The last eight cases were operated at the Presbyterian Hospital, five of which came from the special deafness clinic of Dr. E. Fowler, Jr. Post-operatively, they extend over a period from two to seventeen months. Nine cases are more than one year post-operative.

The requirements for operation are now definitely established on a scientific basis. Good bone conduction with masking if necessary is essential. Bone conduction should not show a loss of more than thirty decibels in the three critical frequencies of 512, 1024, and 2048 vibrations. My first case with masking had an absent bone conduction for the 2048 frequency, 38 decibels loss for the 1024 frequency, and 25 decibels bone conduction loss for the 512 frequency. This patient had no immediate or permanent improvement in hearing, although the fenestra is still partially open. The opportunity for hearing improvement seems to decrease as the bone conduction in the 2048 frequency decreases below 30 decibels.

The air conduction loss as an indication for operation varies depending upon the type of deafness present and upon the condition of the middle ear, the Eustachian tube and the nasopharynx. Most patients with less than 35 to 40 decibels loss can adjust themselves to this defect in hearing. Many are improved with the new therapy directed to the nasopharynx, such as the mechanical removal of adenoids, X-rays, and radium therapy, etc. If the hearing loss is unaffected by treatment then operation is indicated.

X-ray findings as to the anatomical arrangement of the mastoid cells have afforded no contra-indication to operation in my experience. The attic space may be small but not sufficiently small to prevent the complete dissection necessary. Active middle ear congestive changes characterized by a retracted drum associated with an injected or reddened handle of the malleus and Shrapnell's Membrane should have the nasopharynx investigated thoroughly for chronic inflammatory changes. These changes do not contraindicate the operation but hearing results will be more permanent if the nasopharynx is kept free from chronic irritation.

The operative technique as a whole can be acquired by sufficient cadaver dissection under adequate super-vision. The technique was successfully carried out in all of my cases to the most minute detail. Every step is essential if one is to cover the fistula with a thin flap in a one-stage operation. I have had no experience with open uncovered fistulae, but observations lead me to believe they will not be successful. What constitutes a proper fenestra surgically is still an unsolved problem. The tendency is to make gradually larger fenestrae. The larger the fenestra, the greater and more permanent is the sensitivity of the fenestra. This is determined by the degree of the nystagmus produced by pressure exerted directly upon the fenestra. A large fenestra is also necessary to remove the over-hanging endosteum particularly on the solid angle side of the bony semicircular canal wall. The endosteum on the inner or solid angle margin is removed because the membranous canal occupies the convex portion of the bony canal. Nager's work on labyrinthine fractures indicates that the periosteal and endosteal layers both lay down new bone in healing, consequently, it is essential that as much endosteum as possible be removed. New bone grows directly on and not out from the endosteal layer. Nager and others showed that the endochondral layers of bone do not regenerate bone. Consequently if an elongated gap is made through the endosteal layer and this opening is sealed with a thin flap of epithelial tissue, it is more likely to remain patent. In a group of my earlier cases where the endosteal layer was uncovered but not bruised or torn there was consistent improvement. This group had open fistulae in five consecutive cases. When small endosteal openings were made, hearing results were unexpectedly poorer than when the endosteal layer was left intact. When a larger opening was made and a greater portion of the endosteum was removed, the hearing results were much better. The fistulae following this procedure remained much more permanently hyperactive. In my whole series of eighteen cases, the fenestra is widely open in nine cases, partially open in three cases, and closed in six cases.

The immediate hearing improvement is usually excellent on the operating table, provided proper pre-operative indications have been met. This improvement usually represents a moderate whisper heard from 5 to 10 feet. The hearing improvement in the first four to six weeks after operation is usually 15 to 35 decibels in each of the three frequencies; 512, 1024, and 2048. In five to eight weeks, if the fistula is closing, the hearing acuity and fistula reaction gradually diminish simultaneously.

Permanent results in the restoration of practical hearing seem to depend to a large extent upon the degree of the patency of the fenestra. The efficiency of this opening is determined by the amount of nystagmus and vertigo obtained when direct pressure is made on the fenestra.

Holgrem, however, thinks that the hearing results are not determined so much by the type of fistula made as the acoustic condition of the labyrinth itself at the time of the operation. This differentiation should be determined

and may be important. The bone conduction tests may not give us an accurate estimate of the viability of the acoustical labyrinth. This may be demonstrated however, when there is a discrepancy between the masked and unmasked bone conduction. Unmasked bone conduction gives one the viability of both labyrinths which is more efficient than one labyrinth. This is comparable to the difference in vision of both eyes over that of one eye. Islands of decreased bone conduction can be demonstrated in some cases with the masker which are not present in unmasked bone conduction cases. This can be elicited only by a critical observer. It will be overlooked by the average technician. This may indicate a potentially weaker acoustic apparatus and will not withstand the trauma of a fenestration operation as well as a labyrinth which is more intact. Evidence of this phenomenon has been observed by myself with the Western Electric 6A audiometer.

I shall now refer to a group of five patients all operated upon consecutively over one year ago who have widely open fistulae associated with much improved hearing. The endosteum was exposed widely in these cases through a large fistula but was left uninjured and unopened. One of this group has lost half of her improvement although the fistula is still very active. There was no particular variation between the unmasked and masked bone conduction in these cases. Later in a following series of cases, I began to perforate and remove some of the endosteum with poorer results in hearing and with more closed fistulae. One case particularly, who had an open fistula seven months post-operatively has lost two-thirds of her original gain. This patient had two islands of absent bone conduction in the 1024 and 4000 frequencies. In spite of larger fistulae and partial removal of the endosteum, my results were not as good as they were when I left the endosteum intact.

Observations, however, led me to believe that the removal of still more endosteum would lead to more active fistulae. The technique of making the fistula larger plus the removal of more endosteum has been carried out in my last three cases with much better results. The first case performed by the latter method has retained a hyper-sensitive fistula for six months associated with a hearing improvement of 30 decibels in 2048, 35 decibels in 1024, and 30 decibels in 512. This patient has a permanent restoration of practical hearing. The second case was performed five months ago and has retained a hypersensitive fistula associated with a hearing improvement of 15 decibels in 2048, 30 decibels in 1024, and 12 decibels in the 512 frequency. The last case operated by this method two months ago still retains a hyper-sensitive fistula associated with a hearing improvement of 27 decibels in the 2048 frequency, 33 decibels in the 1024, and 15 decibels in the 512 frequency.

The hearing improvement in cases with open fistulae and undisturbed endosteum is much less than those where endosteum is removed by approximately 12 decibels. The following table illustrates this point in decibel improvement. Five cases with undisturbed endosteum in the fistula over one year post-operative.

No.	Patient	Date of Op.	2048 Dcb. Improve.	1024 Dcb. Improve.	512 Dcb. Improve.
1.	D.	10-29-1938	22	10	3
2.	H.	11-11-1938	10	15	13
3.	B.	1-19-1939	10	13	7
4.	N.	1-30-1939	20	15	13
5.	S.	3-18-1939	8	5	15 fistula patent
Three cases with removed endosteum in the fistula.					
1.	W.	9-11-1939 six months	30	35	30
2.	M.	10-10-1939 five months	15	30	12
3.	R.	1-29-1940 two months	27	33	15

Observations made March 18, 1940.

Permanent restoration of practical hearing was obtained in one case where the fistula reaction could not be demonstrated. The external opening is markedly constricted in this, a colored patient, and possibly the exact area for determination of the fistula cannot be found. The hearing improvement is seven months post-operative and is represented by 22 decibels improvement in 2048, 25 decibels in 1024, and 10 decibels in 512 frequency. Became dizzy.

Permanent restoration of practical hearing was obtained altogether in eight cases. Hearing improvement not sufficient for a practical and serviceable level was obtained in five cases. Hearing was not aggravated in any case by the operation. Not in five cases.

The following general conclusions are drawn from my experience with eighteen fenestration operations:—

1. Permanency of hearing improvement depends upon a very large fenestra which will remain permanently hyper-active.

2. Partially active fistulae do not usually result in good practical hearing.

3. The technique of the operation can be acquired by cadaver dissection under proper supervision.

4. Audiometric hearing tests made by all machines vary considerably. We can place reliance only on an average estimation of three or more audiometric tests.

5. After having observed Dr. Lempert's work and results for the past two years, and after having performed the operation on eighteen cases myself, I am convinced that permanent and lasting results of hearing improvement can be obtained with Lempert's fenestration operation, providing each step of the operation is meticulously carried out to the most minute detail with the least amount of trauma and sacrifice of the tissues.

DR. JULIUS LEMPERT: Dr. Campbell argues that it does not matter whether or not you tear the flap, remove or keep the head of the malleus, remove or keep the incus, use or don't use the Shrapnell's membrane, or whether or not there is a history of middle ear infection or a perforation of the drum. He then gives the results in his 22 cases as follows: Four fair results, three poor results and thirteen no results. He reports finally that the last two cases in his series were operated upon five and six weeks ago and he believes that they will be the best of all of his cases. He reports further that in these two cases he carried out my operative technique in every detail and that both show a remarkable improvement in hearing and a tremendously active fistula reaction. Last week, I had the privilege to see the cases of Dr. Campbell in Philadelphia and I congratulated him upon his last two cases. They are beautiful, both of them.

I went to Chicago two weeks ago to see the cases of Dr. Shambaugh, Jr. I saw about eight or nine cases that morning and each showed a most beautiful result and was technically perfect. Also the response to the fistula test was very active in these cases. I congratulate him. I also feel that his device for the removal of bone dust is worth trying by every man doing this work. Bone dust is one of the factors responsible for new bone regeneration and I believe with Dr. Shambaugh that the constant flushing of the field by means of his apparatus will help greatly to remove this otherwise disturbing factor.

Last Sunday I had the pleasure of spending the day in Toronto and of examining Dr. Sullivan's patients. I was extremely happy to see the most beautiful results in a number of his cases. Everyone of them was technically perfect, with fistulae wide open and marked hearing improvement. I want to congratulate him here, as I did in his office.

Dr. Greenfield was kind enough to let me see his patients and even to bring some of them to my office yesterday and the day before. Among the latter

were three patients who had been operated upon at the Presbyterian Hospital. They were patients from the otosclerosis clinic of Dr. Fowler, Jr., on Dr. Kernan's service. These three cases all had marked improvement in hearing. Two of them had wonderfully patent fistulae; one had a marked contraction of the external auditory canal, making it difficult to demonstrate the fistula test. Dr. Greenfield showed me other patients also and those cases I saw were all beautifully done and showed good results. I want to congratulate him.

I want to thank the Chairman of this Section and the Chairman of the Philadelphia Society. I want to thank everyone of the members of both Sections and everyone of you ladies and gentlemen here tonight.

I also want to make one further statement. My patients are all private patients. I have no hospital service and I never had one. Nevertheless, I invite and welcome anyone who wishes to see my patients at any time, at my office or at my private hospital. Anyone who wishes to observe my operative work is also welcome. My bad results and good results are always open to every otologist who wishes to see them, and I am extending the invitation here tonight. Thank you very much.

Patient: Mrs. J. A., age 26 years. Operation: March 17, 1939. Complaint: Progressive loss of hearing five years, dating from birth of child. History: Family trait, no previous history of any ear discharge. Examination: Explanatory audiometric charts.

Patient: Miss A. L., age 24 years. Operation: December 16, 1938. Complaint: Progressive loss of hearing both ears, more marked in right. History: No previous aural infection: family trait. Examination: Explanatory audiometric charts.

Patient: Miss J. J., age 21 years. Operation: February 1, 1939. Complaint: Progressive loss of hearing both ears, more marked in right. Rapid change within last three years. History: Family trait. No previous history of any ear discharge. Examination: Explanatory audiometric charts.

Patient: Miss A. V. D., age 35 years. Operation: December 4, 1939. Complaint: Progressive loss of hearing both ears. More marked in right ear. History: No previous aural infection. Family trait. Tinnitus occurred before hearing impairment. Examination: Explanatory audiometric charts.

Note: The latter two cases are both graduate nurses.

RESULTS OBTAINED WITH THE LEMPERT FENESTRATION OPERATION FROM JULY, 1938, TO AUGUST, 1939.

Patients operated	9
Fenestrum open, with positive fistula test and good improvement in hearing (17 dcb)	1
Fenestrum partially closed with negative fistula test and fair improvement in hearing (15 dcb)	1
Fenestrum open but case not suitable for operation	1
Fenestrum open but hearing improvement lost after 6 months	1
Fenestrum closed, no improvement	6
Postoperative serous labyrinthitis	1
Total ears operated in 9 patients	11

EVALUATION OF THE LEMPERT FENESTRATION OPERATION.

1. In what proportion of cases is the operation successful?
2. How permanent is the hearing improvement?
3. How great is the hearing improvement? Will not a properly fitted hearing aid provide as much hearing without the risks of surgery?
4. What are the hazards of the operation (a) to the ear? (b) to the life and health of the patient?

RESULTS OF LEMPERT FENESTRATION OPERATION ON 18 PATIENTS

	Case	Ear	Date of Operation	Immediate Result (Average of three conversational frequencies)	End-Result
1	Mr. GTC	Left	7-13-38	+17 decibels	Fenestrum closed. Improvement lost.
		Right	8-16-38	+12 decibels	Same as left.
	(Revision)	Left	1-24-40	+35 decibels	+35 decibels.
2	Miss MC	Right	7-20-38	+17 decibels	Fenestrum open. Improvement lost 6 mos. after op.
3	Mrs. BP	Right	7-27-38	Serous labyrinthitis. No gain.	No improvement. Neg. fistula test.
4	Miss AK	Right	8-17-38	+8 decibels	Fenestrum closed. Improvement lost.
5	Mr. PLJ	Right	10-12-38	No improvement. Case not suitable for operation.	Fenestrum open.
6	Mrs. EL	Right	10-26-38	+18 decibels	Fenestrum closed. Improvement lost.
7	Miss KF	Left	11- 9-38	+38 decibels	Fenestrum closed. +10 decibels.
		Right	1-10-39	+23 decibels	Fistula test gone (but fenestrum not quite closed when revised. +15 decibels (Fair).
	(Revision with constant irrigation)	Right	4-23-40	+30 decibels	Fenestrum open. Total gain +47 decibels. (Excellent)
8	Miss PS	Right	7-13-39	+20 decibels	Fenestrum open. +17 decibels. (Good)
9	Mrs. JEV	Right	8- 9-39	+20 decibels	Fenestrum closed. Improvement lost.
10	Mrs. CHA	Right	11- 2-39	+27 decibels	Fenestrum partially closed. +8 decibels.
11	Mr. RT	Left	11-22-39	+20 decibels	Fenestrum open. +17 decibels. (Good)
12	Mrs. GKH	Right	1- 3-40	+20 decibels	Fenestrum open. +17 decibels. (Good)
13	Mr. WJ	Right	1-17-40	+12 decibels	Fenestrum partially closed. Improvement lost.
	(Revision with constant irrigation)	Right	5-15-40	Too recent	Fenestrum open.
Constant irrigation during fenestration:					
14	Mr. CFM	Right	2- 7-40	+30 decibels	Fenestrum open. +32 decibels. (Excellent)
15	Mr. WP	Left	2-21-40	+12 decibels (Serous labyrinthitis)	Fenestrum open. +12 decibels. (Fair)
16	Miss ED	Left	2-28-40	+30 decibels	Fenestrum open. +20 decibels. (Good)
17	Mrs. CVM	Right	3-12-40	+32 decibels	Fenestrum open. +32 decibels. (Excellent)
18	Mrs. EBV	Left	4-24-40	+25 decibels	Fenestrum open. +28 decibels. (Excellent)

Summary: 18 patients, 20 ears, 23 operations (3 revisions)
14 patients with fenestra open
12 patients with improvement in hearing, averaging +24 decibels for the conversational frequencies for those who have gone long enough to be regarded as permanent

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLARYNGOLOGY.

Regular Meeting, Nov. 20, 1940.

Surgical Aspects of Carcinoma of the Larynx. Dr. John D. Kernan.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Roentgenotherapy of Cancer of the Larynx and Pharynx. Dr. Maurice Lenz.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Lantern Slide Demonstration of Portmann Operation. Dr. George R. Brighton.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

(Continued from May issue.)

DISCUSSION.

DR. JOHN D. KERNAN: I would like to state my position about X-ray a little more clearly. There is no doubt that X-ray will cure early cordal lesions and will cure them perfectly, as you saw in these two men presented tonight. But — it is expensive, it is painful, and in my experience it is not only uncomfortable for a few weeks but for six months or longer. On the other hand, I feel this way about it: If you had been presented with the proposition of laryngectomy, say, 30 or 40 years ago, and had been told that the patient would have two chances out of a hundred of recovering, you would have thought it was a poor operation. Now, when the question of laryngectomy is brought up, you can say that the mortality will be less than 5 per cent. I think the improvement in radiotherapy in the last 10 years has been so great that we ought, as honest practitioners, to give it every chance we conscientiously can, and I personally intend to do it. On the other hand, we know what cordectomy will do but we don't know what radiotherapy will do. As far as surgery versus radiotherapy for extrinsic tumors is concerned, I can't see radiotherapy myself. The results are poor anyway, very poor.

I think Martin and I agree perfectly about almost everything. He can't pick a quarrel with me, or get me mad on the question of my definition of intrinsic and extrinsic. He said that when any tumor starts intrinsically, it is still intrinsic even if it spills over the edge of the pharynx. Certainly if the cat gets out of the kitchen window it is not still in the house! I wish to emphasize this point: once an intrinsic carcinoma appears outside the larynx it actually takes on a new nature. Seemingly, Dr. Martin does not believe this.

DR. JULES WALTNER (by invitation): I have had occasion to assist Dr. Portmann in Bordeaux in 25 cases where he used his new method of laryngectomy, and I am glad to confirm the good results illustrated by Dr. Brighton. In these 25 cases, Dr. Portmann did not lose one case from postoperative complications, and the pharyngostomy made it possible in many cases to operate on carcinomatous extensions into the lateral wall of the pharynx, which it was not possible to approach with former methods. In every case, Dr. Portmann performed a tracheotomy; he stressed the great importance of a tracheotomy just over the sternum, performed at least two weeks before laryngectomy. We get a large area of intact skin between the suprasternal orifice and the upper operative wound; descending infection of the lower respiratory tracts is, therefore, excluded.

I should like to ask Dr. Brighton if in these four cases of his, tracheotomy was performed in all or only in one case.

DR. GEORGE R. BRIGHTON: We did a low tracheotomy in only one case before operation.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLARYNGOLOGY.

Regular Meeting, Dec. 18, 1940.

Hemangioma of the Antronasal Sinus Successfully Treated by Radium.
Dr. Samuel Morse.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. WALLACE MORRISON: Hemangiomata of the mucosa of the nasal cavities and the paranasal sinuses are rare, but since they produce such marked symptoms a number have been reported. The most common location is the so-called bleeding polyp of the nasal septum. Less common is the location of the growth in the ethmoid cells or maxillary sinus. All are smooth or papillary, very bright red masses of variable size but usually not larger than a pea, which bleed profusely, spontaneously or on slight trauma, as in blowing the nose; they always bleed when touched with a probe. This is diagnostic when one considers that the tumor is also soft and yielding on palpation, as opposed to the other type of bleeding tumor which may have a similar appearance — carcinoma — but which is firm.

These growths are benign, but cause serious anemia when they bleed freely and often. They may enlarge slowly but they do not infiltrate, and they cause no metastases. Biopsy may be necessary to establish an accurate histologic diagnosis but it is usually attended by severe hemorrhage.

A single case of Dr. Lee Hurd, in which the growth, the size of a small raspberry, occurred on the floor of the maxillary sinus, was diagnosed by noting that the repeated bleeding came from the natural orifice of the antrum, and the growth was found on performing a radical antrum operation. It was not visible in a sinus radiograph. Instillation of a radiopaque medium such as would be used nowadays would no doubt have revealed a small filling defect.

Where the tumor is accessible, as on the nasal septum, it may be removed surgically by excision, including a good margin of mucosa about the base. Where the mass lies in the ethmoid cells, as in the present case, radiation therapy is undoubtedly the treatment of choice. In the maxillary sinus, the external approach allows of complete removal. Electrocoagulation of the mass is not to be advised, as it is almost surely to be followed by very severe hemorrhage when the slough separates.

DR. BABCOCK: I relinquished the chair because I wanted to discuss this case. I think there is no question that chemotherapy is the answer to the recovery in cases like this. We have tried all the other procedures mentioned and — I was going to say 99 per cent, but at least 98 per cent, die. With chemotherapy the deaths reported have dropped to 35 per cent, and I think it is essentially the chemotherapy which causes recovery. I would be very pleased if anyone else had any opinions to offer on the subject.

DR. GEORGE H. O'KANE (by invitation): The case I had in mind, and I have treated several of them since, was one of multiple hereditary telangiectasia. I don't think it would be advisable to treat hemangioma as we did those cases; namely, by injections of quinine and urethane. One of my colleagues had a case of hemangioma with hemorrhage which was treated in this way and the results were not too happy, but in Osler's disease we have had three or four cases which have done very well. The small discrete telangiectatic lesions found in the mucous membranes in Osler's disease can be safely obliterated by quinine and urethane 5 per cent solution, whereas a hemangioma with its rich vascular channels presents the danger of severe hemorrhage, which is

best avoided by the use of radium, as has been demonstrated in Dr. Morse's successfully treated case. For those of you who may be interested in the injection treatment of Osler's disease, may I say that I have reprints of my original article available which I shall be glad to mail on request.

Type III Pneumococcus Meningitis and Septicemia with Complete Recovery. Dr. M. Moghtader.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. M. MOGHTADER: I think the pathway of infection in this case was through the bone and possibly the lateral sinus, as it was found infected. As far as nicking of the dura is concerned, many cases have been reported by Dr. Cunning, and I think that considerable drainage is obtained by nicking the dura in the middle fossa, especially when the foot of the bed is raised.

Of course, I can't say definitely what part of the treatment was responsible for recovery in this case, because we had to do everything possible in order to save the man's life. Whether the final good result was due to sulfapyridine or nicking of the dura and removal of foci of infection, it is hard to tell. In many other cases, most of these procedures have been performed, and no good results have been reported.

(To be continued in a succeeding issue.)